

THE EFFECTS OF DISEASE ON INSECT POPULATIONS¹**EDWARD A. STEINHAUS²****INTRODUCTION**

MOST PAPERS and treatises dealing with the dynamics and natural control of insect populations treat only superficially, if at all, with the effects of infectious disease on such populations. In the literature on the biotic factors known to influence insect populations, the phrase "parasites, predators, and disease" is a common one, but the attending discussions are usually concerned with the parasites and predators while the effects of disease are usually ignored or treated lightly. This neglect is not necessarily the fault of the particular author or investigator concerned since, in truth, he has available to him very little information on which he can rely or from which he can draw definitive conclusions. With few exceptions, hard, cold facts and profound basic knowledge concerning the role of disease in the ecology of insect life are lacking. Nevertheless, even in the absence of the desired large body of well-grounded facts, it might be profitable to examine the areas in which we do possess creditable knowledge, that we may better orient our thinking in preparation for a concentrated attack on the problems involved. The present paper therefore is offered with the hope that it may constitute at least a hesitating step in the direction of a fuller appreciation of the important role of microbial pathogens of insects in the population dynamics and general ecology of insects.

Although the fact that insects are susceptible to disease was known and recorded before the Christian era, the reducing and regulatory effect of diseases on insect populations was not significantly noted until the last half of the nineteenth century. During this period, the isolated observations on entomogenous fungi and the classical work of Pasteur (1870)³ on the diseases of the silkworm set the stage for an increasing awareness that insects of many kinds were subject to attack by disease. LeConte (1874), Metchnikoff (1879), Prentiss (1880), Krassiltschik (1886; 1888), and Brongniart (1888) in Europe, and Hagen (1879), Snow (1890), and Forbes (1895, *a, b, c*; 1896) in the United States were among those who, before the turn of the

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³ See "Literature Cited" for citations, referred to in the text by author and date.

century, either observed outbreaks of disease in populations of insects or advocated the artificial distribution of disease agents as a means of suppressing damaging numbers of insect pests. L. O. Howard (1897) was among those who first accorded disease its place as a distinct and significant member of the family of biotic factors that influence insect populations when he recorded the fact that under certain circumstances "disease steps in" and reduces the population to a point below its normal level.

To be sure, during this same period, and earlier, the effect of disease on populations of vertebrate animals was also being observed and reflected upon. Malthus (1798), the founder of population studies, while developing the idea that populations tend to increase geometrically while the "means of subsistence" (chiefly the food supply) increases arithmetically, concluded that pestilence, along with famine and war, was one of the natural factors that checked the numerical increase of organisms. In his *Origin of Species*, Darwin (1859) refers briefly to the association between the inordinate increase in numbers of a species and the frequent appearance of epidemic disease. The periodic occurrence of disease in populations of rodents and other wildlife (as in voles; Elton, 1936) has been known for many years. Experimental epizootics of bacterial and viral infections among mice and other laboratory animals have been studied in considerable detail by Topley (1926*a, b*); Greenwood, Hill, Topley, and Wilson (1936); and others. Much has been learned from these studies that can be applied theoretically to epizootics among other animals, including insects. And, finally, the conception that infectious disease among humans may be thought of from an ecological and population viewpoint has had increasing realization in recent years as evidenced by the writings of such medical scientists and epidemiologists as Maxey (1948), and Burnet (1953).

With the turn of the century, entomologists, and others, progressively realized that a better understanding of the dynamics of insect populations would enhance the effectiveness of man's efforts to control destructive pests. Ecologists realized that many of the more profound problems in their science could be attacked only on the basis of population studies. In addition, theoretical biologists and mathematicians assumed an intense interest in the subject. Thus there developed several schools of thinking that sought to explain or clarify the principles of population dynamics.

The chief theories of natural control, from the earlier, more narrowly conceived theories, to the later, more comprehensive conceptions, have been reviewed by Solomon (1949). He classifies these theories as: (1) the early "biotic" theories (Escherich, 1914, 1924; Howard and Fiske, 1911; Friedrichs, 1927); (2) the competition theory (Nicholson, 1933; and, to some extent, H. S. Smith, 1935); (3) the "physical" theories (Bodenheimer, 1928, 1930, 1931; Uvarov, 1931); (4) the theories of periodic fluctuation, including: a) the overpopulation theories (Eidmann, 1931, 1934; Dymond, 1947; Kalabukhov, 1935; and others), b) enemy-prey oscillations (Lotka, 1925; Volterra, 1926, 1931; Nicholson and Bailey, 1935), and c) the meteorological theory (Elton, 1942); and (5) comprehensive theories (W. R. Thompson, 1929, 1939; Schwerdtfeger, 1941). (See also Franz, 1950; and Thalenhorst, 1950.)

In the paragraphs that follow, we shall endeavor to examine the few existing theories or concepts relating to the phenomenon of disease as it affects populations of insects, to review briefly some of the literature in this regard, and to attempt to provide provisional answers to certain questions concerning the characteristics of disease as a factor in natural control. Original laboratory and field observations made by members of our own laboratory staff will also be used to aid in the discussion.

DISEASE DYNAMICS

The Epizootic Wave

Fundamental to the proper consideration of our subject is some understanding of the nature of epizootics and the so-called "epizootic wave" (or, as some prefer to say "epidemic wave"). The words "epizootic" and "epizootiology" (analogous to "epidemic" and "epidemiology" in the case of human diseases) are widely used in the literature pertaining to diseases in animal populations. An *epizootic* disease is simply one in which there is an unusually large number of cases; that is, it is a disease or a phase of a disease of high morbidity and one that is only irregularly present in recognizable form. An *enzootic* disease is one that has a low incidence but is constantly present in a population. The current trend among epidemiologists (for example, Maxcy, 1948) and others is to employ the terms "epidemic," "epizootic," and "epiphytotic" for diseases among humans, animals, and plants, respectively. In entomology there is an additional cogent reason for preferring the use of "epizootic" to that of "epidemic" for diseases among insects in that the latter word is regularly used to indicate the presence of or outbreaks of large numbers of insects without regard to their state of health. Thus, although in many respects having a meaning similar to that of "epidemic wave" in human diseases, the term "epizootic wave" is appropriate in referring to this phenomenon in diseases of insects.

In the epizootiology of diseases among insects we attempt to explain infectious diseases of the animals on the basis of mass phenomena; that is, we are concerned with diseases as they occur in groups of insects rather than in the individual insect. Thus, the number of insects afflicted by a given disease and the manner in which this number increases and decreases in a given period of time are the important attributes that characterize the epizootic wave. This wave, a variation in time, usually takes the form of a single-humped curve with ascending and descending arms of almost equal or of different lengths. That is, the epizootic wave may express itself in one of three different forms: (1) with a longer descending than ascending arm; (2) with the arms of equal or nearly equal lengths so that the bell-shaped curve is symmetrical or nearly symmetrical; and (3) with a longer ascending than descending arm.

Since the agents responsible for disease maintain themselves in nature between epizootics, the above curve is actually but a small part of the greater curve that would result if the interepizootic periods were plotted. Between epizootics the pathogen may exist in a free-living phase or in a non-active, latent, or resistant stage; or the disease may manifest itself in the

form of an enzootic. Sooner or later, however, its activities may be enhanced to a point where an epizootic ensues. When this happens the number of insects afflicted with the disease will increase, sometimes slowly, sometimes rather rapidly, and the curve will rise correspondingly to a peak representing the maximum number of animals affected at any one time. When the peak is passed, the number of diseased insects then falls with varying degrees of rapidity back to zero or to the normal enzootic level. When the numbers or percentages of affected insects are plotted against time, we have a curve representing the epizootic wave.

The period just preceding the increase in numbers of affected insects (that is, the increased prevalence of the disease), is known as the *preepizootic phase*. During this phase the epizootic potential of the infecting micro-organism may be rising as the result of an increase in virulence for the insect. This may come about in various ways, a common one that of the virulence being enhanced by the repeated natural passage of the pathogens through successive host individuals. On the other hand, environmental or intrinsic influences may play upon the susceptibility of the host, thus causing the insect to be less resistant to attack by the pathogen. A greater degree of dissemination and an increase in numbers of the infecting agent usually take place during the preepizootic phase, as does an increasing rate of transmission from infected insects to healthy ones. There may also be a rising dosage of pathogens received by the insects in the particular population concerned. In most instances the density of the susceptible population during the preepizootic phase is of considerable importance. If the density is low or if only a few insects are present, the disease is likely to die out, or remain enzootic, before it has a chance to enter the epizootic phase. On the other hand, if the population density is high, the preepizootic phase may end rather abruptly and be of short duration, with a sudden rise in mortality initiating the epizootic phase.

The *epizootic phase* itself is the climactic portion of the epizootic wave. Much of what transpires during this phase is inadequately known as far as insect diseases are concerned. We are continuously tempted (and indeed we yield to the temptation) to adopt some of the principles learned from the studies of epizootic waves among vertebrate animals and of epidemic waves in human populations. There is, of course, considerable danger in assuming that what is true concerning diseases in vertebrates is also true of diseases among insects. Nevertheless, in the absence of the desirable amount and kinds of data pertaining to insect diseases, an expedient alternative is to make analogies from the data and concepts furnished by studies of the effects of disease on populations of higher animals. In some instances this information is derived from experimental as well as from natural epizootics. The knowledge thus gained can be augmented, corrected, or replaced by the increasing amount of data and basic information that are accumulating from the study of epizootics among insects *per se*. In the discussion that follows, the circumstances just mentioned will be taken into account, and a special effort will be made to present and emphasize the data and information, inadequate as they are, available from observations of epizootics in insect populations.

It is perhaps apropos at this point to emphasize the great need in insect pathology for intensive investigation in the realm of what might be called "experimental epizootiology." Such inquiry would, in a sense, parallel the research of Topley (1926*a, b*); Greenwood, Hill, Topley, and Wilson (1936), and others in the study of experimental epizootics of bacterial and virus infections among mice and other small animals. Undoubtedly it would also include various biomathematical considerations. As indicated in the preceding paragraph, although much can be learned from the study of diseases in vertebrates and applied to insects, such analogies cannot be carried very far nor used safely as a basis of prediction. Differences in such things as mechanisms of innate resistance, effects of temperatures on homoiothermic vertebrates as contrasted with poikilothermic insects, as well as other physiological, anatomical and ecological differences greatly limit, for the insect pathologist, the value of data obtained from the experimental study of diseases in vertebrates. Accordingly, the experimental study (including the use of appropriate statistical methods) of epizootics among insects, as an adjunct to the study of epizootics as they occur in nature, is long overdue and worthy of much time and effort. Such a study is required to obtain the accurate and reliable data essential to the formulation of principles pertaining to the epizootiology of disease in insect populations.

The *postepizootic phase* of the epizootic wave is characterized by the nature and number of the survivors. As with most forms of life suffering disease or disaster, at least a few surviving insects remain to perpetuate the species. The number of survivors varies with each epizootic, but in general it would appear to be determined largely by chance or by factors not yet ascertained or understood, as well as by the variable original susceptibilities of the insects making up the population. The role of acquired immunity, important in epizootics among vertebrates, is not well enough known in natural epizootics among insects to permit generalization. The degree of dispersion of the survivors among fresh hosts and the effect of this on the proportion of subsequent survivors are likewise not well known in the epizootiology of insect diseases.

Since the epizootic wave represents a variation of disease prevalence in time, it is appropriate to inquire as to whether or not these variations assume any particular pattern. Considering the epidemiology of diseases occurring among human populations, Stallybrass (1931) grouped the variations in the prevalence of disease under four headings: (1) short-time irregular fluctuations; (2) annual seasonal variations; (3) cyclic or intrinsic periodicities; and (4) secular variations. Substantial data pertaining to epizootics among insects are available for only the first two of these categories. Short-time irregular fluctuations may result from a variety of causes, such as variations in temperature, humidity, nourishment of host (qualitative and quantitative), population densities, intercurrent infection and parasitism, and such man-made factors as applications of insecticides. Annual seasonal variations are those that are dependent more or less directly on the seasonal meteorological changes. The effect may be on the pathogen itself, on the host, or on both. The high prevalence of certain insect diseases may be observed at particular seasons of the year, depending on the climatic character of the

geographical location concerned. Also, the annual seasonal variation may be related to an annual build-up of the insect population and to the seasonal development of certain broods or stages of the insect. Cyclic or intrinsic periodicities of duration in months and years but not coinciding with the annual solar cycle may occur but few confirmed observations of the phenomenon have been reported. It is true that some insect diseases appear to flare up in epizootic form every so many years, but it is not clear that these outbreaks represent actual cyclic periodicities. Perhaps the clearest example of this type of relationship is that between a pathogen and an insect having a life cycle of several years (for example, a seventeen-year locust and a fungus disease). Secular variations, that is, those observed only at great intervals, such as once a century, have not been competently observed as far as insect diseases are concerned. One might conceive of a hypothetical case in which an insect is introduced into a new locality without one of its diseases; a secular period of time might elapse before the disease "caught up" with its host.

It is, of course, obvious that the effect of disease-producing microorganisms on populations of their hosts should always be considered in relation to the attributes of the insects themselves, and to such factors as insect parasites and predators, climate, and other environmental agencies. However, inasmuch as physical factors, such as climate, influence primarily the degree of the effect of the microorganisms rather than the kind of effect, and since disease may act on insect populations in the absence of such biotic agencies as insect parasites and predators, the discussion in this paper is limited to a consideration of those factors concerned directly with the occurrence of disease in insect populations, and with the related epizootiological aspects.

Any epizootic affecting an insect population is concerned with three primary natural entities: (1) **the infectious agent**; (2) **the insect host**; and (3) **the environment**. Each of these factors has certain attributes that when properly related to the attributes of the others play their appropriate role in determining the initiation, rise, and decline of an epizootic.

The Infectious Agent

At least three attributes characterize microbial agents capable of causing disease epizootics in insects and thereby reducing insect populations. These are:

(1) **The capacity of the agent to invade and infect the host insect.** This, as we have indicated, is dependent upon certain attributes of the host that concern the insect's susceptibility or resistance to the pathogen. From the standpoint of the microorganism, however, its capacity to produce an infection or disease in a given insect is determined largely by its ability to produce toxins, enzymes, and other anabolic substances, and to bring about the mechanical destruction of cells or tissues. The pathogenicity or virulence may vary considerably with the particular strain of microorganism involved, and this variation may occur during the course of the epizootic, during the enzootic period, or in a free-living state. The virulence of some

strains may be enhanced by repeated passage through susceptible hosts or by mutation. Conversely, the diminution in virulence frequently takes place when the strain is associated with a resistant host, or during a free-living period, or through mutation. It is important to remember that in appraising the effects of a disease agent on a population of insects, the invasion or infection of the host by certain highly virulent pathogens almost always results in the death of the host (as in some virus infections); or the pathogen may be so benign that only a chronic disease results and the population is only gradually or almost imperceptibly depressed (as with certain gregarine or coccidian infections).

The ability of a pathogen to infect an insect may also be related to dosage. In some instances only a few microorganisms are necessary to bring about an infection, whereas in other cases a relatively large number are required. A "critical dose" is necessary to overcome the innate resistances offered by a particular insect to a particular microorganism.

(2) **The capacity to survive.** This may be a matter of a microbial species being naturally long-lived (because of innate morphological, physiological, or ecological qualities), or it may be that through the mechanism of spores, cysts, or resting cells, the microorganism is capable of surviving for long periods of time in the midst of environmental conditions ordinarily destructive to its vegetative stages. Some microorganisms are unable to serve as efficient biological control agents because they tend to die between the time they are applied and the time they are ingested or otherwise contacted by the susceptible insect.

(3) **The capacity to spread.** The capacity of a pathogen to spread from one insect host to another is one of the most important factors concerned in any epizootic. Involved in determining this capacity are the various natural methods by which microorganisms are transmitted to healthy insects; these will be considered in some detail in a subsequent section of this paper.

The Natural Distribution of Entomogenous Microorganisms

The natural distribution of microorganisms associated with insects varies according to a number of factors. Those microorganisms that are obligate parasites or symbiotes of insects have their distribution governed by that of their insect host. Free-living microorganisms that may also be part of an insect's regular microbiota, and those microorganisms that are associated with insects in a purely fortuitous manner, usually are widely distributed. From an ecological viewpoint the geographical limitations to the distribution of such species may be conveniently and broadly classified into terrestrial, marine, and fresh-water forms. The almost world-wide distribution of many of the free-living microbes is made possible by the fact that "microbial environments are microenvironments, hundreds or even thousands of which lie concealed from the gross ecological eye in any gram of soil" (Stanier, 1953).

Nevertheless, the environmental limits of geographically widely distributed microorganisms are frequently very narrow. The ecological niche may be so constituted that the occurrence of the proper conditions of nutri-

tion, temperature, and humidity may be limited to but a few millimeters, thus confining microorganisms to an exceedingly small area. Now, because the environmental limits of most microorganisms are so narrow, a microorganism and its special microenvironment are not always in contact. And since most microorganisms have short generation-times and produce large populations, it is logical that when a particular microenvironment occurs (as in the lumen of an insect's alimentary tract), it will be rapidly occupied by the corresponding microbial species. Should the microenvironment be removed, the marked capacity of dormancy possessed by many microorganisms enables the microorganism concerned to survive until the recurrence of the microenvironment, provided it recurs before the limit of dormancy is reached.

The fact that in the insect gut, for example, we frequently find two or more species of microorganisms flourishing "side-by-side" does not, of course, mean that these microorganisms are occupying the same ecological niche. The several species comprising such a localized group of microorganisms each derives its own mosaic of nutritive requirements from the substrate on which they are subsisting (the ingested food of the insect), and similarly exploits the remainder of the precise constellation of environmental factors that is required by it. To be sure, the presence of even a single microbial species in the intestinal tract of an insect may so completely dominate the microenvironment as to destroy the niches of other microorganisms that are ingested by the insect. This type of domination undoubtedly is one of the principal factors that determine the number of microorganisms regularly found in the gut of an insect. Other factors, such as the liberation of toxic metabolic products, may also be involved in determining the kind and number of microbial species associated with insects.

Any present-day attempt to describe the geographical distribution of those microorganisms known to be pathogenic for insects is destined to be frustrated by the lack of adequate data. Those microorganisms that are obligate pathogens of insects will, of course, be found nowhere except in areas covered by the distribution of their hosts. Thus, *Bacillus larvae* White, the cause of American foulbrood in the honeybee, has been found only where honeybees occur. Similar examples may be cited from among the fungi, protozoa, and viruses pathogenic for insects. To be sure, the host distribution range is nearly always greater than that of the pathogen so that the distribution range of the latter cannot be said to be identical with that of the host. This is the case whether the pathogen occurs in a single host species or in several.

When we consider the facultative pathogens, however, we are virtually lost when stating their geographical distribution. Those pathogenic bacteria and fungi that are able to thrive saprophytically as well as in the insect body may have a geographical distribution far more extensive than that of the insects subject to their attack. The distribution of even obligate pathogens may be limited by environmental factors—for example, areas where the insect abounds may be free of the pathogen (in certain fungi, for instance) if the humidity and temperature required by the pathogen are

never appropriate (for example, in areas where the climate is suitable for the insect but too hot and dry for the fungi).

The Adaptation of Microorganisms to Insects

In considering the role of the infectious agent as one of the natural entities concerned in any epizootic affecting an insect population, it is pertinent that we have some idea of the adaptive or evolutionary processes by which there arose microbial pathogens capable of causing disease in insects. Are the processes essentially those of gradual adaptation to a state of pathogenicity, or were they those of genetic mutations?

As has been pointed out by Stanier (1953) in another connection, microbial populations have peculiarities which, measured against the norm of animal populations, have bearing on the problem of adaptation of microorganisms to their hosts. In the first place, because of the very short generation time of microorganisms, the total number of individuals comprising many microbial species is enormous when compared with the numbers of its host species. Thus, the gut contents of but a few individual insects may contain as many or more representatives of the microbial species as the total number of representatives of the insect species concerned. Secondly, some microbial populations have a great capacity for dormancy. Although the characteristic resistance to adverse environmental conditions exhibited by the sporeforming bacteria is well known, that many nonsporeformers are capable of surviving in a nonproliferating vegetative condition for long periods of time is also true (for example, see Steinhaus and Birkeland, 1939). This long survival time is especially striking when measured against their extremely short generation time. Relatively long periods of dormancy are also characteristic of many fungi, protozoa, and viruses that affect insects.

The adaptive capacities of microorganisms, especially bacteria, are known to be extremely great. Consider, for example, the ability of certain bacteria to use organic compounds unknown in nature and synthesized only in the laboratory. The adaptive capacities of microorganisms are further emphasized by the ability of certain strains of bacteria, for instance, to become resistant to and actually feed upon such protoplasmic poisons as hydrocyanic acid and carbon monoxide, and upon such therapeutic agents as penicillin, streptomycin, and the sulfonamides. Nutritional sources available to microorganisms cover the range from completely inorganic compounds to the most complex of organic molecules. The organic compound that cannot be used as nutrient by one or more microorganisms probably does not exist in nature. They survive and grow in a wide range of conditions with optimum growth rates being reached, sometimes very rapidly in nutrients with which they were unfamiliar and in which the initial growth may have been meager. Incalculable numbers of niches thus are occupied by microorganisms with their omnivorous habits and almost ubiquitous occurrence. It is not surprising, therefore, that microorganisms of all major types long ago probably became adapted to life in association with insects.

The environment, as it affects living organisms, undergoes two principal kinds of variation or change: a systematic variation, usually gradual, uni-

directional, and long ranged in character; and a fluctuating variation of short-term duration that fluctuates about a mean, and that may be of the nature of a cyclic event (for example, seasonal changes) or of a purely random event. It is on this basis that Stanier (1953) has suggested the term *evolutionary adaptation* in referring to an organism's adaptation to systematic changes, and the term *physiological adaptation* in indicating adaptation to fluctuating changes:

Evolutionary adaptation constitutes the moulding of the genotype of an organism through evolutionary history by selection to fit the mean conditions of its environment. . . . Physiological adaptation . . . If it represents a response to a cyclic event which has a periodicity much longer than the life span of an individual organism, it *may* involve the alternate selection of different genotypes, and thus have a purely genetic, even though evolutionary static, basis. . . . Random fluctuating variations, on the other hand, occur constantly over periods that are short in comparison to the life span of the individual organism, and in such cases adaptation always involves direct phenomic accommodations to the change.

Now, how can these concepts be applied to our knowledge of microbe-insect relationships? In the first place, it is rather obvious that the obligate gut, caecal, and intracellular symbiotes have been associated with their insect hosts for great periods of time. In all probability the same may be said concerning those microorganisms that are a regular and more or less constant part of the gut microbiota, as well as those plant and animal pathogens requiring an insect vector, and those microorganisms pathogenic for their obligate insect hosts. In each of these instances the microorganism has met with the systematic variations of the environment and has undergone evolutionary adaptation to a greater or lesser degree. On the other hand, the microorganisms that are associated with insects in a purely adventitious or fortuitous manner, and some of those which are found in the insect's alimentary tract as a result of the insect's momentary diet (for example, saccharolytic bacteria in the presence of a diet high in sugars), are examples of microbe-insect associations based on fluctuating variations and represent instances of physiological adaptation.

Just what factors have been instrumental in determining which microorganisms were to adapt themselves to living with which insects can only be guessed. The systematic changes in microenvironments may occur over a much shorter span of time than they do in macroenvironments. Inasmuch as microorganisms have such an extremely short generation time, evolutionary adaptation can take place much more rapidly than with higher plants and animals, even insects themselves. Whereas centuries would be involved in the study of evolutionary adaptation in elephants, the development in natural populations of strains of bacteria resistant to certain sulfonamides and antibiotics has occurred in less than a decade. Experimentally, in pure cultures, microorganisms have been induced to undergo evolutionary adaptive changes even more rapidly. In the case of microorganisms associated with insects, it is conceivable that certain anatomical locations available to microorganisms in the body of the arthropod may be of such a character as to isolate the microbial clone concerned from the many selective forces that affected it in nature; this reduction in the number of variables, and the exposure to the selective forces provided by the insect (together with the

more precise physicochemical environment provided by the insect), would in all probability enhance the opportunities for the microorganism's adaptation to life in the insect.

Just when and how the first microbial pathogens for insects arose and became "adapted" to their arthropod hosts is a conjectural matter. It is probable that the pathogenic habit of entomogenous microorganisms has multiple origins. Most authorities conceive of it as developing from monophagy, from commensalism, or from mutualism. Others think it represents the extension of an initial encounter between the host population and a population of microorganisms. Many believe pathogens are too specialized to be capable of departing from the pathogenic or parasitic adjustment once it is established, although the tendency of such microorganisms to evolve toward mutualism is recognized. That the many intracellular symbiotes occurring in numerous species and groups of insects at one time possessed pathogenic or invasive powers appears to be somewhat more probable than that these mutualistic symbiotes are evolving toward virulent pathogens. In either case arguments could be advanced that the adaptation was in keeping with the main evolutionary trend which appears to be one of progressive loss of synthetic function. Obligate parasitism or pathogenicity and obligate mutualism both are examples of specialized conditions of growth in which a loss of synthetic ability is indicated, and either may be an evolutionary advantage to the microorganism.

Without the presence of definitive data, there has been some justification in assuming that the ability of microorganisms to invade and cause disease in insects came about through evolutionary stages that gradually approached the parasitic or pathogenic habit. A hypothetical sequence of such evolutionary steps could include those beginning with autotrophic forms of microbial life (although direct evidence of such derivation is scanty), through the free-living microorganisms thriving on dead organic matter, then the putrefactive microorganisms living in the alimentary tracts of insects, to those microorganisms that live freely in the gut or body cavity of the insect but affect their host primarily by means of soluble exotoxins, and finally to those that may not only be capable of producing toxins but also of invading healthy tissues and causing disease and pathology in them.

It is frequently assumed that a high degree of pathogenicity in an organism indicates a recent and still imperfect host-parasite relation; that hosts and parasites that have lived together for a long time develop a toleration for each other. That such is always the case has been questioned, and Ball (1943) attributes pathogenicity to a kind of innate incompatibility between the parasite and hosts. According to this author:

...evolution may, in many cases, have brought about a mutual adaptation between host and parasite resulting in relative harmlessness of the relation, but in other instances no such decrease in pathogenicity seems to have occurred; and in still others as the parasite becomes better adapted for life in its host, it has become rather more or less capable of producing disease.

As far as the diseases of insects are concerned, no well-authenticated examples have been presented to support either of these concepts. From the meager evidence at hand there would appear to be merit in Ball's con-

tention that the development of a tolerance is not always the rule. Nevertheless, one cannot escape the feeling that in most cases a pathogen and host do tend to develop a more or less balanced relationship when, as described by Allee (1949), for example, the association has been a prolonged one, when there has been an opportunity for a high proportion of the host species to become infected, and when there is no substantial means by which the pathogen can survive for long periods in the absence of the host.

In light of the tendency of animal hosts to tolerate their pathogens after they have been associated for long periods of evolutionary time, it would seem that the possibility of a commensal or a mutualist evolving gradually into a virulent pathogen does not materialize as frequently as may have once been supposed. Nor would it appear necessary that a pathogen always represents a slow evolutionary adaptation to its host, or that low pathogenicity indicates a recent association with no evolutionary adaptation. Although there are a few noteworthy exceptions, most of the present microbial pathogens of insects are not of the type ordinarily found in commensal or mutualistic associations with insects. Instead of a gradual adaptation of these types of entomogenous microorganisms to a pathogenic role, it would seem more likely that virulent pathogens arise through genetic mutations. If the mutant is not preadapted to its host or if it has no common evolutionary history with its host and comes in contact with it suddenly for the first time, it may rapidly die out (or, possibly mutual elimination or disoperation may occur). On the other hand, if the proportion of resulting disease in the total population is not great, and satisfactory adaptation occurs, the mutant may thrive in the susceptible population, and gradually the insect may develop a high degree of toleration to the pathogen that initially exploited it.

The Insect Host

The second primary factor with which epizootics are concerned is the insect host and its susceptibility or its resistance to the attacking microorganism. An insect may be (1) an occasional, (2) a principal, or (3) an obligate host to a particular species of microparasite or pathogen. Which of these it is depends, of course, not only on its own extrinsic and intrinsic properties, but upon those of the pathogen and the environment as well.

If a particular pathogen is entirely dependent upon a particular insect species (or group of species) for its survival, it must be continuously successful in gaining entrance into the insect's body, in reaching the particular tissues and anatomical sites that favor its multiplication, in making an exit or departure from the host, and in surviving in an extra-insect situation for a time sufficient for it to reach or be contacted by a fresh, susceptible host. Examples of obligate insect pathogens that meet these requirements abound: the entomophilic Entomophthoraceae, the bacteria causing the foulbroods and those responsible for the milky diseases, entomophilic Microsporidia, and the insect viruses, and others.

When a microorganism is less dependent upon a particular insect or group of insects for its survival, and is able to utilize other hosts or survival mechanisms, it need not be continuously successful in progressively meeting

the four critical requirements just mentioned. Thus, the insect host is no longer an obligate one but may still be the principal host or only an occasional one. Thus, it would appear that certain insects are the principal animal hosts for *Bacillus thuringiensis* Berliner which also is capable of living apart from insects. Certainly the primary habitats for such common bacteria as *Aerobacter aerogenes* (Kruse) and *Serratia marcescens* Bizio are not insects; yet, on occasion, these "saprophytes" are capable of invading and producing fatal diseases in certain insects. To some extent this depends on characteristics of the microorganisms themselves, but it is certain that attributes of the host are also involved. To what extent, therefore, is the insect as a host responsible for the propagation of the pathogen and for its maintenance and accessibility in insect populations? Why are some insects highly susceptible to a given microorganism while other, even closely related, species are virtually refractory to it? Why is a given insect population highly susceptible to a certain pathogen at one time, and much less so at another? What are some of the intrinsic factors that determine the susceptibility or the lack of it for a particular insect species? These are some of the questions that come to the minds of nearly all students of insect diseases and their effect on insect populations. Unfortunately, only fragmentary and incomplete answers can be provided at this particular stage of our knowledge.

Let us briefly examine the information we do have for whatever value it has in helping us to understand the role of the host in the epizootiology of insect disease. In the first place, insects, like all other living things, are insusceptible to the great majority of microbial life they contact during their life span. Most microorganisms coming in contact with an insect or being ingested by it are simply adventitious bits of life without any predilections whatever toward interfering with the life processes of the insect. Such microorganisms may pass on through the insect none the worse for their journey, others succumb to the digestive processes of the insect, and still others may find the animal's gut and gut contents to its liking and remain, for a longer or shorter period, as an integral part of the insect's microbiota. Most microorganisms coming in contact with the external surfaces of the insect merely assume the role of epicoles (epibionts) or phoretics.

This ability of the host to remain refractory to the activity of most of the microbial life it contacts is usually attributed to what is called its "innate immunity," its "natural immunity," or its "resistance." Certain aspects of this form of immunity or resistance have already been referred to as representing a lack, on the part of the microorganism, of evolutionary adaptation, or, in some cases, an evolutionary adaptation of the host toward a toleration of the pathogen. It is an immunity in which specific humoral antibodies play virtually no important part. Instead, it is based on the natural protective properties of the animal's anatomy, physiology, biochemistry, and genetic make-up. A discussion of the role of these factors in innate immunity has been presented elsewhere (for example, Redaelli, 1929; Paillot, 1933; Steinhaus, 1949); suffice it to remark here that although this type of immunity or resistance expresses itself in numerous ways, from so-called phylogenetic resistance to external mechanical defenses, the manner in which it actually functions in insect populations is known largely

by inference and assumption. Nevertheless, in any thorough study of an epizootic one must recognize that the disease may be fatal to one species of insect but harmless to another species feeding on the same host plant, or that one stage (or age) of the host insect may be highly susceptible to a disease while another stage of the same insect is entirely resistant, or that the particular structure or mechanical arrangement of an insect's mouth parts may preclude its contacting or ingesting a pathogen that gains ready entrance to the alimentary tract of an insect with chewing mouth parts.

The relation of inadequate nutrition to the occurrence of disease in individuals and populations is well known among humans. Elton (1942) and others have made similar correlations in rodent populations. The weight of evidence seems to indicate that in general a favorable food supply, that is, adequate nutrition, may aid an insect in resisting microbial invasion; a reduced food supply, that is, inadequate nutrition, may enhance susceptibility, encourage debility, and increase mortality. When food is scarce, most animals tend to move about over larger distances in search of food; this increased movement may promote the spread of organisms the activities of which add to the mortality. To what extent such relationships exist in insect populations is not known; remarkably few studies (as, for example, the one by Schwecowa, 1950) have been made in connection with insects. Indeed, there exists extremely little reliable information relating to how nutrition affects the resistance and susceptibility of individual insects or small groups of insects to disease, let alone large populations. The literature does, however, contain a number of papers that speculate on the matter.

Largely innate but not wholly so (there is some evidence that insects may be stimulated to produce opsonins) is the protection afforded the host by virtue of its phagocytic tissues—that is, cellular immunity. The role of the blood cells, or hemocytes, and the pericardial cells, in enabling an insect to withstand certain types of microbial attack, has had a fair amount of study but no one has ventured to evaluate its importance from the standpoint of its protective influence in a population of insects vulnerable to microbial attack.

Another type of immunity believed to manifest itself in insects results from the activities of true antibodies and is known as "acquired immunity" since it is acquired by the animal during its lifetime. There appears to be no doubt but that individual insects are capable of producing antibodies of a kind when stimulated by the appropriate antigen. A number of workers have claimed to impart an immunity to certain insects against certain bacterial pathogens by injecting the arthropods with appropriate vaccines or other antigenic preparations. It is noteworthy, however, that there has as yet been no convincing demonstration of immunity, based on the presence of humoral antibodies, in insects collected in nature, to a pathogen to which the insects were ordinarily susceptible. The main reason for this deficiency is probably largely because very few attempts to accomplish this have been made. That acquired immunity is of considerable significance in the epizootiology of vertebrate animals, there is no doubt. That we are safe in assuming that the same holds true in insect populations is open to question. To be certain one way or the other, it is essential that we examine more care-

fully the nature and status of survivors of insect populations that have suffered epizootics.

In the case of vertebrate animals, survivors of one epizootic may be present in the replenished population that is struck by the next epizootic. This is not usually the case with the survivors of an epizootic in a population of insects. By the time an epizootic has run its course the surviving insects have usually completed their metamorphosis, migrated, or died from other causes. In the meantime, a new nonimmune population has arisen and the influence of any immune survivors that persisted may fade into insignificance. To be sure, this is not always the case (as has been observed with some stored-grain insects and certain long-lived species, and in possible instances in which the "resistance" is carried over to the next generation), but with most of the insects infesting field and truck crops, forests, and ornamentals, it tends to hold true.

Among the factors, other than immunity and resistance, that determine the course of an epizootic, and that may be attributed to the host, are those that may be grouped together under the term "host behavior." To some extent this designation includes certain aspects of population densities, which we shall consider shortly, but it applies directly to such matters as the spatial distribution of the host, its feeding habits, the manner and degree of its movements, and the like.

It is obvious that insects which regularly space themselves at relatively great distances, or which, because of low population density, are "few and far between" in a given area, are less likely to contact pathogens arising from other individuals of its species than if they were in close proximity of each other. Direct contact between healthy and infected (or dead) insects is an important means of spreading many entomogenous fungi (because the dead insects may bear infectious conidia externally), and an important factor in numerous diseases that may be acquired through the cannibalistic or "nipping" habits of insects. Direct contact of a healthy insect with regurgitations or with anal exudations of an infected insect may occur but usually such discharges spread the infectious agent by contaminating the food plant that will be ingested by healthy individuals.

The tendency of some insect species to aggregate and others to disperse may be an important factor in determining the severity and extent of a disease, but very little experimentation and few observations have been made concerning the matter. One may assume, however, that in those instances in which the spread of an infection is dependent largely upon the movements of diseased individuals among healthy ones, the maximum opportunity for the spread of a disease will occur when a center of close aggregation is associated with marked dispersal. In other words, infected insects moving out radially from a node or center at which the disease is fulminating, enable the disease to spread on contact more rapidly and more thoroughly than if such movement (that is, dispersion) did not occur.

Other types of movement by the host insect are also important. In the case of certain polyhedroses, for example, the infected insects tend to move to the tops of the host plants or trees. In this location they hang by their prolegs and die. The disintegrating bodies of the insects contaminate the foliage

beneath them, or are washed down by rain, and the spread of the virus is thus enhanced. Some diseases are spread by flying insects, the dispersion of the insects usually taking place before the disease has progressed to a point at which the insect is no longer able to fly. An interesting variation of the latter is a fungus (*Massospora cicadina* Pk.) infection of the periodical cicada, *Magicicada septendecim* (Linn.). Unlike most entomogenous fungi, *Massospora cicadina* produces conidia within the body of its host. The insect's abdominal segments are eventually sloughed off, exposing the conidial mass. However, the insect is still able to move and fly about, and these movements of the insect aid in scattering the conidia of the fungus.

The dispersion and migration of diseased insects apparently conform to the general principles governing the dispersion and migration of healthy insects, as reviewed by Wolfenbarger (1946) and others. In the absence of exact data, however, it might be logical to assume that the extent of such movements would, in most instances, depend on the degree to which the insects were infected. In the very early stages of disease the insect's own movements would hardly be affected, whereas as the disease progressed the insect would be rendered incapable of extensive, spontaneous movement until just before death, when it would be incapable of progressive movement of any kind. Insects being dispersed passively or by mechanical means would be less likely to have their movement affected by their diseased state except as the advent of the insect's death may alter the situation.

The feeding habits of most insects are, of course, determined by a number of intrinsic factors. Regardless of this, the end result may be one that endangers or protects the insect. As we have already indicated, one of the reasons why insects with sucking mouth parts are subject to relatively few bacterial and protozoan diseases undoubtedly is because there is little opportunity for microorganisms to gain entry into their digestive tracts during the feeding process. Those insects with chewing mouth parts are more likely to ingest food contaminated with pathogenic microorganisms. This is especially true for bacteria, protozoa, and viruses, for which the portal of entry is usually the mouth and digestive tract. Although some fungi do infect insects via the digestive tract, the characteristic portal of entry is through the body integument. There may also be some association between the type of food an insect eats and its propensity to contact disease organisms. We have elsewhere (Steinhaus, 1953a) commented on the interesting fact that most of the insects suffering from polyhedrosis virus infections feed on the open leaves of the host plant. This, however, may be purely coincidental since there are a number of exceptions.

It is of interest to note that some scavenger insects, such as the cockroach, rarely suffer from highly fatal diseases even though their feeding habits should provide ample contact between them and microbial pathogens. A close study of their intestinal microbiota, however, reveals that these insects frequently do suffer from numerous "inapparent" infections. Parasitic protozoa that exert but slight deleterious effect on the host are commonly found in the gut lumen or in the intestinal epithelial cells. Furthermore, it appears logical to assume that during the long evolutionary development of these insects, there developed, through the process of selection,

innately resistant strains capable of fending off lethal attacks by microorganisms repeatedly encountered in their scavenging. To be sure, however, there are examples in which this manner of conjecture does not hold up.

In any case, the picture as it relates to insects as a whole, although filled with many apparent inconsistencies, does seem to indicate that those insects ingesting their food by the chewing process are considerably more prone to more types of disease than are those insects (along with mites and spiders) that obtain nourishment by the sucking process. One must be cautious, however, not to extend such a generalization too far. The correlation may be largely coincidental. In the case of mosquitoes, for example, the larval stage, which takes in its food by means of chewing (that is, opened mouth parts) suffers from a greater number of infectious diseases than does the adult. Here, however, the aquatic environment of the larva, as opposed to the aerial one of the adult, may have as much, or more, to do with the prevalence of infection as does the difference in feeding habits. In other adult-larva comparisons (for example, in *Lepidoptera*) the adult may be innately resistant to the infection (for example, by certain viruses) regardless of the type of mouth part. As to the actual factors involved in this matter, a safe generalization might be the following: (1) the innate resistance of one stage (or host species) to an agent to which another stage is susceptible (for example, adult *Lepidoptera* compared with larvae); (2) the ability or habit of an insect to feed on a food source in a manner that avoids contamination and infection (for example, many *Homoptera-Hemiptera* in their use of piercing-sucking mouth parts); and (3) the protection afforded certain insects by their behavior and environment (for example, certain leaf-mining insects, especially those that spend their entire larval life in one leaf).

Thus far we have been dealing briefly with the first two (infectious agent and insect host) of the three primary natural entities with which any epizootic is concerned. The third of these entities—the environment—will be considered in its various aspects in the section to follow.

CHARACTERISTICS OF DISEASE AS A FACTOR IN NATURAL CONTROL

As has been the case with most branches of science, insect pathology has passed through those early phases during which our subject (that is, the phenomenon of disease in insect populations) was a matter of mere idle curiosity to the observer. In time, this curiosity became intensified and gradually took the form of a more penetrating inquiry as to the nature of the phenomenon as far as natural control was concerned. Finally, the subject is beginning to receive not only careful attention but diligent scientific investigation. Even those entomologists, and other biologists, not directly involved with diseases of insects have, at one time or another, given the subject enough thought to be interested in at least some aspect of it. Indeed, it has been the author's experience that most of these workers, as well as those biologists more directly interested in the subject, characteristically ask insect pathologists a pattern of questions that fairly well

penetrate to the heart of the matter as far as understanding the role of disease in the natural control of insects is concerned.

Most of the questions pertaining to the characteristic of disease as a factor in natural control may be arbitrarily grouped so as to be covered by about six main statements of inquiry. Both for the purpose of presenting the available information in a manner that will conform with these fundamental questions, as well as for reasons of convenience, the discussion to follow will be arranged somewhat in the form of question and answer, with the realization that such a division of the subject matter is somewhat artificial and that, in reality, the contents of all questions and all answers are closely interrelated. In the discussions to follow it is our intention to quote frequently from the writings of numerous authors in order to state controversial viewpoints accurately and to present positions and trends of thought without loss in shades of meaning.

1. To what extent and in what manner is disease a density-dependent factor?

As distinguished by Howard and Fiske (1911) and W. R. Thompson (1928), and emphasized by H. S. Smith (1935), the mortality factors affecting a population of insects may be considered as *density-dependent* (that is, factors that kill a greater percentage of insects, the greater the population density), and *density-independent* (that is, factors whose effect is not related to the population density). The principal density-independent factors are climatic; the chief density-dependent factors include natural enemies, and, when limited, food supply, space, protective niches, and the like. According to this concept, disease-producing microorganisms, along with insect parasites and predators, are considered as natural enemies, and hence as density-dependent mortality factors. They are density-dependent factors because they affect a greater proportion of the insects as the population density increases. This, in turn, is the result of the fact that the denser the population the more easily does transmission from the environment or from diseased to healthy individuals occur, and under these circumstances the microorganisms are able to increase at a greater rate and in greater proportion than the increase of the host insects.

As far as disease is concerned, it is to be assumed that density-dependent and density-independent factors are both involved in the totality of epizootic dynamics. As Eckstein (1939) and others have pointed out, climatic conditions and the nature of the environment in which an insect lives may affect or determine not only the degree of susceptibility of the insect or the virulence of the pathogen but the population density itself.

That the relation between the population density of insects and the outbreak of disease was noticed several centuries ago is indicated by the chronicled warnings of the dangers that result from the overcrowding of silkworms during the rearing of these insects for silk production. Today the dangers (due to disease) of crowding are widely recognized in the rearing of many insect species. During the latter part of the nineteenth century similar observations were reported in the case of insects in nature. Infection by entomogenous fungi, such as the renowned chinch bug fungus, was early considered to be "dependent upon the abundance of the host" as well as upon

favorable weather conditions. By the early 1900's the association between numbers of the host insect and the occurrence of disease became a generally accepted fact, as evidenced by this statement of Howard's (1902):

It has long been known that when grasshoppers appear in enormous numbers they are apt to die off as a result of some apparently contagious fungous or bacterial disease, and not only are grasshoppers affected in this way, but other insects as well when they swarm in enormous numbers, the chinch bug, for example.

Other examples of the relationship between host abundance and disease incidence abound. To Howard and Fiske (1911), for example, disease did not appear to become a factor of natural control until a density level had been reached that made "the insect in question, *ipso facto*, a pest." These workers also believed that only through parasites and predators is a pest to be brought under complete natural control, "except in the relatively rare instances in which destruction through disease is not dependent upon superabundance." Morrill and Back (1912) found that the efficacy of fungi in destroying whiteflies under natural conditions was dependent upon the abundance of the insects, and that a period of "excessive abundance always precedes effective temporary control." (According to Fawcett 1944, however, some fungi are only partly favored by a high whitefly density.) Glaser (1915), who made many of the early observations in eastern United States on the "wilt disease" (a polyhedrosis) of the gypsy moth caterpillar, stated: "Epidemics of disease occur only in localities heavily infested by the gypsy moth." Conversely, in lightly infested woodland "the caterpillars are much more widely separated and an epidemic is not produced." Speare (1922), noted that the period of maximum abundance of young citrus mealybugs in Florida orchards is likewise the period in which the percentage of diseased specimens increases the most rapidly. Dustan (1924*b*; 1927), on the basis of his study of a fungus disease of the European apple sucker, asserted that the abundance of the host insect was one of the three factors (the other two being adequate humidity and high temperatures) upon which the outbreak of epizootics is almost entirely dependent. A similar opinion has been expressed by Cox, *et al.* (1943) in their report on a fungus disease of the Comstock mealybug, as well as by numerous others. Růžička (1924) recorded that as populations of the nunmoth caterpillar in European forests built up over a three-year period, a polyhedrosis virus disease had its greatest incidence during the year of greatest host density, but that in areas of low population density the disease was also present and effective in reducing the numbers of insects present. Similar observations have been made by others who have studied epizootics in forest insects. Bird (1948), for instance, in discussing the conditions under which a polyhedrosis of the European spruce sawfly is most effective in reducing the host population, states: "The only factor necessary for its ability to destroy the insect is a sufficient number of the host in the forest and this number has been found to be quite small. . . . The disease has remained effective at low levels of sawfly population." Bird points out, as do Thompson and Steinhaus later (1950) in another instance, that those conditions which are favorable for the development of the sawfly (that is, encourage high population densities) are also

favorable for the development of the disease, although at times a factor such as malnutrition may play a role in promoting susceptibility.

Thus, the evidence that disease is more prevalent or more noticeable in insect populations of high density than in those of low density is seen to be plentiful. Accordingly, there can, in general, be no quarrel with the idea that certain of the effects of pathogenic microorganisms are dependent upon the density of the host population. Especially is this true in the case of naturally induced epizootics. As indicated by Bird's statement quoted in the preceding paragraph, however, it does not necessarily follow that populations of low density do not occasionally have a high percentage of their number affected by disease. Much may depend on the degree and nature of the exposure of the susceptible insect to the pathogen. One Japanese beetle grub in a soil plot heavily charged with spores of the bacterium responsible for milky disease (that is, with spores deposited by a previous generation of diseased grubs present in large numbers) will very likely acquire the disease as soon as it would if hundreds of grubs were present. Of course, this may be the reflection of an earlier high density of the insect concerned. The effect of a mortality factor need not be dependent solely on the immediate population density of the host, but is often dependent on the population density of the preceding generation. It is perhaps logical to expect this phenomenon to occur frequently in the case of disease organisms, such as sporeforming bacteria, that can survive over relatively long periods of time.

An epizootic that breaks out when the density of the host population is high may gain a momentum that extends it for long distances regardless of the density of the population. Thus Balch and Bird (1944), while recognizing that there is probably a minimum level of population in which the disease could maintain itself, nevertheless observed a polyhedrosis of the European sawfly to extend into areas of light infestation and, from the standpoint of natural control, to be "remarkably effective" under such conditions. At the University of California, Clark and Thompson (unpublished observations) noticed that in the case of tent caterpillars, the "momentum" of a virus-caused epizootic may extend a disease in time as well as in space. A "heavy" epizootic one year may result in a highly diseased population the following year, and possibly longer, even though the second population may be relatively small.

Furthermore, that epizootics can be initiated in populations of low density when the infectious agent is artificially distributed has been recognized since Reiff, in 1911, reported that "In selecting the localities in which the disease ['wilt disease'] is to be introduced, it is unimportant whether the caterpillars of the gypsy moth are present in large or small numbers." Relatively recently, it has been clearly demonstrated in the use of a polyhedrosis virus in the control of the alfalfa caterpillar (Steinhaus and Thompson, 1949; Thompson and Steinhaus, 1950). It is in such situations that we may consider disease not so much as *dependent* upon a population of high density as *enhanced* or *accelerated* by the high density. On the other hand, the fact that a disease operates at a low host density does not mean it is density-independent. It should be remembered that when man introduces or applies the infectious agent it creates an artificial situation that may be of little

value in judging whether or not the agent acts on the population in a density-dependent manner.

It appears clear that disease is one of the "overpopulation phenomena" that develop when a population is able to increase in density rapidly or over a period of years until a level is reached at which a disease suddenly appears and soon reduces the population to a low level. This is followed by a disappearance or relaxation of the disease and another period of increase in the population. This effect of disease has for some time been recognized in populations of small vertebrates, such as voles (Elton, 1936), hares (MacLulich, 1937), ruffed grouse (Clarke, 1936), and others. (See also Dymond, 1947.) In the case of insects, the literature contains numerous references to disease being one of the factors responsible for the "crash" following the gradual increase in numbers of the species concerned.

Certain of the virus diseases show rather clearly that epizootics may be primarily responsible for the cessation of the density increase and the "crash" of the insect population. In 1944, for example, Balch and Bird reported the place of a polyhedrosis in the natural control of the European spruce sawfly, *Gilpinia hercyniae* (Htg.). When first discovered in Canada in 1930, and for the following eight years, only rare individuals that might have been diseased were observed in the forest. During the latter part of 1938, numerous diseased larvae were noticed in some parts of New Brunswick, and Dowden (1940) observed them in heavily infested areas in Vermont and New Hampshire. The disease spread over wider areas and by 1940 was considered to have been responsible for controlling the outbreak in Vermont and New Hampshire. By 1942 it caused a "striking reduction" in populations in Canada. In 1942, in Maine, where it was observed in 1940 (Peirson, 1941), the disease apparently had killed from 63 to 98 per cent of the dead larvae beaten from trees in studied plots (Dirks, 1943). Balch and Bird concluded that density of the sawfly populations seemed to be the most important factor determining the control effect of the disease, and that although it proved remarkably effective under conditions of light infestation, there was no doubt that the percentage of disease increased with the numbers of its host. This increase appeared to be independent of secondary effects of crowding, such as the shortage of food, and there was no evidence that the disease was greatly influenced by weather conditions.

The logistic curve and formula for population growth depict the fact that as density increases a point is reached at which the trend will be reversed and the rate of increase will begin to decline. Finally, a density level is reached beyond which the population density never passes. It is generally assumed that this phenomenon is brought about in two ways (see Solomon, 1949): through new and more severely unfavorable processes coming into play at successively higher levels of density, and by an increase in the intensity of action of various individual density-dependent factors or processes. Diseases may function in both of these manners. An example of the first, that is, processes unfavorable to the increase or to the maintenance of the density concerned, was cited as early as 1911 by Howard and Fiske. From their studies on the factors important in the natural control of such defoliating Lepidoptera as the gypsy moth and the browntail moth, these workers

concluded that at moderate densities parasites appeared to be the principal unfavorable factor, that disease becomes effective when an insect has increased to far beyond its average abundance, and that famine and starvation, "the most radical means at nature's disposal," play their subjugating role at the highest population densities. A statement by Solomon (1953) is also pertinent in this regard. According to this author,

Animal populations tend to increase whenever the general conditions are favourable, but are limited at varying levels by natural enemies, disease, food shortage, overcrowding and so on. They are often controlled at a safe economic level by parasites, predators and perhaps by disease. But if these factors fail to hold the density at a moderate level, and the physical conditions are favourable to the species, then an upper limit to abundance is set by the restrictions of space, food supply and cover, typically through competition. At the same time, predators, parasites and particularly disease may make an important contribution to control at these high levels.

One of the most interesting studies of the effect of a pathogen on an insect population is that by Ullyett and Schonken (1940) who were concerned with a disease of *Plutella maculipennis* Curt., caused by the fungus *Entomophthora sphaerosperma* Fres. As is characteristic of entomophthoraceous fungi, *E. sphaerosperma* discharges a ring of conidia about the body of the infected insect on the leaf of the plant. Healthy insects contacting these conidia are likely to become infected. Thus, the more crowded the population, the more frequent are the contacts between individuals, and the greater the chance of the fungus passing from diseased to healthy insect. Ullyett and Schonken make the point, however, that while the *spread* of the fungus among the population of hosts is dependent largely upon the density of the latter, the *appearance* of the disease is wholly dependent upon weather conditions. They conclude, therefore, that, except in special environments, the disease "cannot be regarded as a density-dependent mortality factor in the same class with parasites and predators." They further state:

...in this country [Union of South Africa] at least, fungus disease is entirely dependent upon weather and so may be classed as a density-independent mortality factor. As it cannot, therefore, influence the average population density of its host directly, any such effect must be produced indirectly through its reaction upon the population of natural enemies.

In other words, according to Ullyett and Schonken, some entomogenous fungi act as density-independent factors since regardless of how dense the host population may be, if the weather conditions are such as to make it impossible for the fungus to germinate and grow, the disease will not develop. At this point, however, it is necessary to emphasize the statement that if this generalization were true it would have to be limited to certain entomogenous fungi and would not necessarily apply to other insect pathogens. A common portal of entry for most entomogenous fungi is the integument. Before the fungus spores or conidia can germinate and penetrate into the body of their host, high humidities and adequately warm temperatures are required. On the other hand, with most bacteria, protozoa, and viruses, the portal of entry is the insect's mouth, and once in the animal's gut, humidity adequate for the pathogen's development is automatically provided. Usually, temperatures favorable to the insect are also favorable to the pathogen. For

this reason, as the authors no doubt intended, the conclusions of Ulyett and Schonken, if accepted, should be limited to the particular situation with which they were concerned, and to similar situations in which the initiation of a disease is dependent upon highly favorable weather conditions.

It would appear, however, that a further note of caution is in order since the qualifications we have just attributed to the conclusions reached by Ulyett and Schonken are not adequately reflected in a more recent contribution by Ulyett (1953). This author declares that:

Disease factors are peculiar [as mortality factors in insect populations] and belong to a class of their own. This is indicated by (1) The appearance of the disease among the population is wholly dependent upon the intervention of suitable weather conditions in the environment. . . .

Unfortunately Ulyett does not make clear, if such be the case, that his remarks are based upon a study of certain fungus diseases. Certainly such a generalization cannot be extended to all diseases of insects; the evidence to the contrary is too voluminous and convincing. In the case of fungus infections of the type studied by Ulyett and Schonken, there is little doubt that favorable weather conditions may permit the onset of disease, and once it has appeared, its spread is largely dependent upon host density. However, situations exist (for example, in California) where the weather may at times be favorable for the development of fungi but the insect hosts are not present in sufficient numbers to enable the fungus to operate in an epizootic manner.

Ulyett (1953) concludes that the *Entomophthora* disease in *Plutella* is neither wholly density-dependent nor density-independent, but passes through phases that include both characteristics. Furthermore, he finds the various theoretical explanations relating to the actions of a factor such as disease, or to actions on the part of parasites, to be inadequate to explain "this peculiarity."

Whereas the observations of Ulyett and Schonken (1940) are no doubt reliable, their conclusions, and those of Ulyett (1953), are open to question or reinterpretation, at least as far as certain of their implications are concerned. The fact that an infectious agent must have certain conditions of temperature and humidity before being capable of infecting its insect host does not invalidate the fact that it is density-dependent. The fact remains that if a greater proportion of the host is infected when the population is high than when it is low, the mortality factor is density-dependent regardless of how it gets that way. The action of parasites and predators is also dependent upon favorable weather. If a disease organism is present in an area in which it will operate under favorable weather conditions, it will operate in a density-dependent manner.

It is of interest to note that Müller-Kögler (1941a) studied somewhat similar epizootics in which populations of *Panolis flammea* Schiff. near Leipzig were attacked by the fungus *Empusa aulicae* Reich. This investigator made an effort to evaluate the various predisposing and mortality factors (age of larvae, nutritional deficiencies, weather, population densities, et cetera) involved in the epizootics he observed. Of these, weather and population density appeared to be the most important. However, the weather did

not always appear to play the deciding role in determining the outbreak of the fungus disease. He concluded that probably the population density was of decisive importance and that the disease becomes an epizootic, usually only after the larvae have increased to great numbers.

The effect on insect populations of what might be considered to be more or less chronic infections (that is, diseases that are not highly virulent and that do not rapidly sweep through a population) may be seen from the work of Park (1948) in connection with his study of competition between populations of the flour beetles, *Tribolium confusum* Duval and *Tribolium castaneum* Herbst. Both of these species are susceptible to infection by a coccidian parasite of the genus *Adelina*, presumably *Adelina tribolii* Bhatia. This protozoan, although capable of killing its insect hosts, is not a highly virulent pathogen. Park (1948) and, in a subsequent report, Park and Frank (1950) have summarized their interesting findings essentially as follows:

Uninfected populations of *T. confusum* behaved like infected populations in terms of density and pattern of growth, except that uninfected populations contained about 20 per cent more adults (and 20 per cent fewer larvae and pupae) than did infected populations. On the other hand, uninfected populations of *T. castaneum* were strikingly influenced by the absence of *Adelina*. Their mean density, assayed over a continuous period of 1,140 days, was 33.5 individuals per gram of medium as compared with a density of 13.3 when the protozoan was present. That is, without changing the food supply or otherwise modifying experimental conditions, the equilibrium density increased approximately 2.5 times. As with *T. confusum*, uninfected cultures contained about 20 per cent more adults than did infected cultures. When both species of flour beetles are placed together in competition cultures, the population trend usually is in favor of *T. confusum* if the cultures are infected and in favor of *T. castaneum* if the cultures are not infected. In uninfected populations, *T. castaneum* persists in about two thirds of the cases, instead of in about one tenth in infected populations. A 2,070-day study of the beetles, encompassing about 69 continuous generations, revealed that *T. castaneum* persists at a higher mean density than *T. confusum* when both are uninfected. When *Adelina* was present, *T. confusum* was but slightly affected while the mean density of *T. castaneum* suffered a 60 per cent reduction.

The dependence of the natural outbreak of disease on the density of the host population is not unrelated to the concentration (or population density) of the infecting agent. Theoretically, the number of microorganisms present in the environment of a host population may be so small that disease does not have an opportunity to manifest itself during the time period covered by the host generation concerned. In a sense, this is the converse of situations in which the pathogen is present in large numbers but the density of the host population is so low that the outbreak of disease is not apparent. (A third type of situation is that in which the presence of large numbers of the pathogen—in this case a fungus—in host populations of high density but under environmental conditions, such as insufficient moisture, prevents the pathogen from attacking the insects.)

The effects of varying concentrations of a pathogen may be clearly dis-

cerned in instances in which the pathogen is artificially applied for purposes of biological control. For example, in experimental plots infested with the alfalfa caterpillar, it has been observed (Thompson and Steinhaus, 1950) that above a certain minimum concentration of polyhedrosis virus there is not a great deal of difference in the control effectiveness with varying concentrations of the virus, or the concentrations may be above levels at which the effects show. In cold weather, larvae treated with the heavier concentrations came down with the disease about one day earlier than did those treated with lower concentrations. In his report on the use of a polyhedrosis virus in the control of the European pine sawfly, Bird (1953) says "Mortality from the disease depends on the amount and concentration of virus used, the method of dissemination, and the stage of larval development at the time the virus is applied." He also points out that the rate at which larvae die from the disease depends, within limits, on the amount of virus consumed. In those areas where greater quantities of virus were deposited, deaths occurred more rapidly and higher percentages of larvae were killed.

From all the available evidence it can only be concluded that disease-producing microorganisms, like parasites and predators, are density-dependent mortality factors in the natural control of insect populations. To be sure, disease, like parasites and predators, is dependent upon and influenced by the weather, but this does not make disease a density-independent factor according to the usual meaning of the term. Disease can manifest itself in low populations, just as can parasites and predators, but its fulmination is dependent upon a relatively high density. Epizootics can be initiated in populations of low density when the infectious agent is applied artificially. Unfortunately, there are available virtually no data that can be analyzed with regard to such matters as the change in the rate of spread of a disease with the change in the density of the population.

2. To what extent are disease epizootics and their effects influenced by weather conditions?

As denoted in the preceding section, the importance of weather conditions in influencing the occurrence of disease in insect populations may be considerable. In some instances, temperature and humidity are of great importance in the initiation of an epizootic. In other instances, temperature and humidity have very little direct effect on the course of a disease except as they affect the activities of the insect host. Despite numerous theories, assumptions, and statements concerning the role of these and other climatic factors on epizootics in insect populations, remarkably few intensive field observations or experimental studies have been made in this connection. It should be remembered that although the effect of weather may be great, it does not determine whether or not disease is density-dependent. The limiting or lethal effects of weather, the effects of weather on the course of an epizootic, and whether or not disease is density-dependent in its effects on populations are actually separate and distinct problems.

Most of the generalizations found in the literature attest to the great dependence of epizootics of all types on "highly favorable" weather conditions. In some respects, this has been unfortunate. Tracing back to the

source of these generalizations, we find that almost invariably they originate with investigations pertaining to entomogenous fungi and the diseases they cause. As we have already explained, most entomogenous fungi attack their host through the integument, requiring adequate external humidity or moisture to carry out the process. Most bacteria, viruses, and protozoa, on the other hand, are ingested by the insect, and their moisture requirements are satisfied by the provisions of the insect's alimentary tract or body cavity. Unfortunately, many of the early writers assumed that what was true of fungus infections in insects was true of those maladies caused by other microorganisms. This has led to any number of false conclusions and assumptions (including undue pessimism about the possible use of microorganisms in the biological control of certain insect pests), and it behooves us to attain the proper orientation of the matter insofar as the available facts and knowledge permit. It was in this connection that in the previous section it was pointed out that because a disease requires certain conditions of temperature and moisture this does not invalidate the fact that it is density-dependent.

In considering the effects of weather in the epizootiology of disease, the close interrelation of temperature and humidity must continuously be borne in mind. This relationship may vary considerably, however, with low temperatures (and moderate to high humidities) being entirely compatible with the outbreak of epizootics (for example, see Müller-Kögler, 1941*b*). Another important related factor is exposure, particularly to wind and sunlight. Insect-infested crops on hilltops subject to prevailing winds may be deprived of the beneficial effects of certain fungi that thrive in lowlands protected from the desiccating effect of winds, or in areas protected by windbreaks. Such effects have been noted by several workers (for example, Betrem, 1938; Wolcott and Martorell, 1940; Waterson, 1940). Similarly, the presence or absence of light has been recorded as a factor in some diseases (for example, Pascalet, 1939; Lambourn, 1921).

Most students of insect pathology are aware that the primary factor found to determine the efficacy of the white-fungus disease or white muscardine—caused by *Beauveria globulifera* (Speg.)—in reducing populations of the chinch bug (*Blissus leucopterus* [Say]) in the Mississippi Valley states, was moisture. The presence of adequate moisture, together with optimum temperatures and an abundance of hosts, provided the conditions favoring the outbreak of the disease over wide areas of the host's distribution. It was the realization of this requirement that caused Billings and Glenn (1911) to decri the artificial distribution of fungus spores for purposes of controlling the chinch bug. In their words:

Moisture conditions have much to do with the appearance of... [the] disease in a field; artificial infection nothing... Apparent absence of fungus among chinch bugs in a field is evidence of unfavorable conditions rather than lack of fungus spores.

Similarly, in the case of the natural control of whiteflies, Morrill and Back (1912) point out that the fungi concerned thrive only under suitable conditions of weather. The same applies to the fungi associated with scale insects on citrus in Florida. The fact that Florida has its rainy season during

the warmer months of the year while California's citrus region has its rainy season during the cooler months, undoubtedly explains why fungi are found associated with scale insects in Florida so much more abundantly than in California. It was this same combination of circumstances to which Speare (1922) attributed the fact that the citrus mealybug, attacked by the fungus *Entomophthora fumosa*, was of minor consequence in Florida but was a serious pest of citrus in California.

Undoubtedly, areas of the world that have warm and humid climates are more likely to support a greater abundance of entomogenous fungi than are more arid regions. The numerous species described from the tropics by Petch and others attest to such an assumption. The actual amount of rainfall has been used in prognosticating the probable success or failure of entomogenous fungi in naturally controlling certain insects. Thus, Skaife (1925) maintained that a mycosis of grasshoppers caused by *Empusa grylli* Fres. developed in South Africa only in localities that had a rainfall of over 14½ inches during a six-months' period. Grasshoppers in Montana were affected by the same disease after four days of rain and of humidity seldom below 98 per cent, but when the weather cleared and the humidity dropped on the fifth day, the disease was checked (Parker, 1924; see also Smith, 1933).

The dependence of the development of entomogenous fungi upon warm, moist, or humid conditions has been attested to by numerous investigators, the literature revealing an almost universal agreement on this point although some workers (for example, Müller-Kögler, 1941a) have reported outbreaks of fungus disease during cold-moist periods. Even in those instances in which the fungi develop, although the general atmospheric conditions are relatively dry or arid, the microenvironment is such as to provide the conditions requisite for this development. For example, in studying an infection of the sugar cane mealybug caused by an aspergillus fungus, Speare (1912) observed the disease to be just as abundant in dry weather as in moist. However, as he pointed out, the leaf sheaths of the sugar cane harboring insects collect and retain moisture for long periods of time. Thus the microclimate may be as important as is the general climate.

Seasonal changes in the amount of moisture, as well as in temperature levels, may explain some of the seasonal appearances of certain fungus infections. The fact that the overwintering larvae of some insects are, because of the accompanying moisture, frequently found attacked by fungi has been recognized at least since the time of Lohde's (1872) report on epizootics caused by fungi.

When we consider those diseases of insects caused by microorganisms other than fungi, the picture is not so clear. Although it is generally recognized that in their extremes, elements of weather, such as temperature and humidity, do affect the course of a disease in individual insects as well as its epizootiology, the effects, if any, of the ordinary fluctuations of these factors are not always apparent.

Early observers (for example, Glaser, 1915) of virus infections in insects in the field reported that climatic conditions appeared to bear an important relation to the appearance and course of the disease. Whether the effect

was a direct one on the virus or an indirect one through its effect on the host, was not ascertained and, in most cases, was not pondered. That the effect was not the same in quality or degree apparent in the case of fungus infections was detected by some workers. Thus, Reiff (1911) noted that the polyhedrosis of the gypsy moth caterpillar "seems to be influenced by climate and weather conditions less than any other caterpillar disease." Similarly, Glaser (1915), in studying the same disease, comments: "... there seems to be no correlation between high temperatures and deaths due to wilt." On the other hand, Glaser suggested that humidity might be as important or perhaps more important than temperature in determining the progress of the disease. Fischer (1914) refers to the frequency of the occurrence of polyhedroses during "rainy summers." Similarly, Komárek and Breindl (1924) observed that a polyhedrosis (*Wipfelkrankheit*) of nun moth caterpillars nearly always occurred after rainy weather had begun. (Possibly in instances of this kind the rain aids in the dispersion of the infectious material on the foliage.) Růžička (1924), however, reported the occurrence of epizootics of nun moth polyhedrosis in what he described as dry weather, and a greater distribution and longevity of the virus in dry air than in moist air. He furthermore explained the *Wipfeln* phenomenon of diseased larvae climbing up (or down) a tree by postulating that if the polyhedrosis strikes the larvae when the atmosphere is damp and cold, the insects climb up to escape to a drier layer of air; if attacked when the air is hot, the larvae go down the tree seeking a cooler situation.

The true relation between weather and the occurrence of virus diseases in insect populations was not significantly clarified during the twenty-year period following these early observations. In 1944, Balch and Bird, on the basis of their study of a polyhedrosis of the European spruce sawfly in Canada, concluded that "there is little to suggest that the disease is greatly influenced by weather conditions." This was followed, in 1948, by a more exacting statement by Bird to the effect that those conditions generally favorable for the development of the sawfly are also favorable for the development of the disease. In 1954, he further stated that, within limits, spruce sawfly larvae die more rapidly when fed highly concentrated dosages of virus and die more rapidly when held at high temperatures. However, very high temperatures inhibited virus multiplication, and in the laboratory the larvae did not die from disease when fed highly concentrated dosages of virus and reared at 85° F but did die rapidly when transferred to a temperature of 72° F.

Field and laboratory studies of a polyhedrosis of the alfalfa caterpillar enabled Thompson and Steinhaus (1950) to eliminate temperature, humidity, and climatic conditions in general as direct factors in the initiation of the disease in the field. On the other hand, appropriate weather conditions are capable of inducing an increase in the density of the insect population which, in turn, appears to enhance the outbreak of disease. Furthermore, temperature was found to be an important factor in determining the incubation period of the disease; the length of time required for the disease to run its course will vary with the temperature. Experimentation indicated that within the temperature range in which the host insect is active, suscepti-

bility to infection is independent of the temperature. In general, it was found that the temperature range optimum for the caterpillar includes the range optimum for the virus.

Although carefully prepared studies of the effect of weather conditions on bacterial and protozoan diseases are few, it would appear that, in general, what we have just said of the virus diseases is true for all the major types of diseases other than those caused by fungi.

One of the first known and most publicized of bacterial diseases has been that of grasshoppers caused by *Aerobacter aerogenes* var. *acridiorum* (d'Her.) (= *Coccobacillus acridiorum* d'Her.) d'Herelle's (1911; 1912) original field observations included only scant attention to ecological aspects of the disease. Subsequent workers, however, expressed more concern about the effects of weather on the disease. Not all of their reports are in agreement. In general, however, most observers express or imply the belief that high humidities facilitate the destructiveness of the disease by their debilitatory effects on the insects. Beltrán (1926), for example, says that rain weakens the grasshoppers and makes them more receptive to infection. Heavy rains, however, are believed to clean the foliage of the host plant contaminated with infectious material and thus decrease the incidence of the disease. In any case, most reports suggest that low humidities are unfavorable for the outbreak of the disease. As far as temperature is concerned, most authorities appear to conclude that while high ground temperatures militate against the outbreak of an epizootic, moderately cool to warm temperatures are favorable to the disease. Nevertheless, outbreaks at high temperatures have been reported. Shulguina and Kalinitshev (1927) found that the grasshoppers were most susceptible to the bacterium when the temperatures fluctuated from 12.5 to 23.5° C, and that higher temperatures (33 to 35° C) resulted in a much lower mortality.

Data such as those just quoted are somewhat difficult to interpret; indeed, much of the information provided by those who have written on the "*acridiorum*" infections in grasshoppers would appear not to support broad generalizations. Furthermore, on the basis of our present knowledge, it is difficult to judge just what mechanisms are involved in the outbreak of the epizootics. Once an infective dose of bacteria gains entrance to the body of the insect host there is no apparent reason why high atmospheric humidity would be required for the full development of the disease in the individual insect. Nor is there an obvious reason why low atmospheric humidity should materially affect the pathogenesis of the disease. Similarly, once the bacteria are within the body of the insect, moderately high temperatures should not adversely affect the course of the infection as long as such high temperatures are not in excess of those in which the insect can survive. In fact, one would expect high temperatures to enhance the course of the disease, unless such temperatures actually inhibited the multiplication of the bacteria. One feature, however, must be remembered: high temperatures, accompanied by direct sunlight and a lack of moisture, would very likely tend to kill large numbers of the bacterium (a nonsporeformer) occurring on the foliage of plants contaminated with material from the dead and dying grasshoppers. Thus, although a considerable amount of the transmission of the pathogens

is attributed to cannibalism, that which is dependent on foliage contamination could be significantly retarded or depressed. In any event, we have here an example where the manner in which the disease may progress in the individual insects is not necessarily reflected by the manner in which the disease may manifest itself in the host population.

Theoretically (and probably in most instances actually), weather conditions that are favorable for the insect should be favorable for the disease. The possibility of disease may be enhanced by the favorable effect of the weather directly on the pathogen or on its insect host, or by the fact that favorable meteorological conditions may lead to the development of dense populations. Excessive temperatures and humidities can, when not inimical to the health of the insect, predispose it to infection. One would not, however, expect the initiation of the disease to be as dependent upon such factors as temperature and humidity as upon the factors of adequate dosage, transmission, and the like. On the other hand, warm temperatures should ordinarily cause a faster multiplication of the pathogen in the body of the insect, thus shortening the incubation period and the period of morbidity. Evidence for, but not necessarily proof of, these generalizations is suggested in the studies of the milky diseases of the larva of the Japanese beetle (Dutky, 1940; Beard, 1945), in *Bacillus thuringiensis* infections in the Mediterranean flour moth (Berliner, 1915), and the alfalfa caterpillar (Steinhaus, 1951a, and unpublished data), in numerous examples of miscellaneous bacterial infections observed in our laboratory and in insectaries, and in experimental infections with nonsporeforming bacteria in corn borer larvae (Metalnikov and Chorine, 1928). Heimpel (1954) observed a decrease in mortality (caused by a strain of *Bacillus cereus*) among the larch sawfly when the mean daily temperature remained below 20° C for "an appreciable length of time" (about two weeks). In insectaries, adverse (that is, excessively high) conditions of temperature and humidity are especially likely to be important in predisposing the insects to infection and they also usually enhance the rate at which a disease develops in a group of insects (Steinhaus, 1953b).

3. To what extent is disease capable of decreasing the numbers of insects in a population? In other words, how near to eradication can disease bring a given population of insects?

The manner in which these questions are phrased acknowledges what appears to be a fact, namely, that disease is not known to completely annihilate or exterminate an insect species over any general area of the insect's geographic distribution. As in epizootics among most other animal populations, in the economy of nature at least a few individuals escape destruction and are thus able to conserve the species. (The factors responsible for this tendency of the last remnants of a reduced population to survive unfavorable conditions have been summarized by Solomon, 1949.)

Nevertheless, in the literature one occasionally finds reports of populations of insects being "wiped out," "destroyed," "100 per cent killed," and the like. Read in context such statements are usually not intended to be accepted literally; but exactly what is the status of the populations usually has not been made clear. Also found in the literature are statements, less dogmatic

yet still not precise in meaning, although, in many cases, descriptive and informative. The following are examples: "Scarcely one in a thousand of the vast hosts of young [chinch] bugs... remain alive,..." (Shimer, 1867). (Incidentally, Shimer, in his enthusiasm, said that diseases are incomparably the most important agents in all nature in destroying noxious insects!) "... places infected early enough, always show... [a diminution in numbers] sometimes to 100 per cent" (Reiff, 1911). "In 1888 the chinch bugs disappeared from some of the eastern counties... and Dr. Snow expressed the belief they were carried off by an epidemic" (Billings and Glenn, 1911). "At times [the fungus] is abundant enough to produce a veritable epidemic, after which there is a scarcity of mealybugs" (Speare, 1912). "... artificially induced spring epidemics have resulted in many cases in the practically complete destruction of the larvae..." (Speare and Colley, 1912). "... so widely scattered were the live mealybugs that a collection of representative specimens was made with difficulty... The rapidity with which such diseases spread when once established is remarkable, and it is strange that any insects escape alive" (Speare, 1922). "... an enormous reduction... but a complete extermination does not take place,..." (Glaser, 1915). "... where [the fungus] was abundant almost one hundred per cent control was brought about" (Dustan, 1924*b*). "In Mähren (Brtnice) war Polyedrie im Jahre 1919, Aussterben der autochthonen Raupen erfolgte im Jahre 1921" (Růžicka, 1924). "... populations of the thistle hopper had been almost completely obliterated... It appeared that the epizootic was present and was wiping out entire populations of the thistle hoppers" (Wilbur and Fritz, 1942). "... a diligent search was required to find even single specimens" (Fisher, 1950). An infected culture of Indian-meal moth larvae was "losing its vitality and slowly dying out" (Steinhaus, 1951*b*). The "... virus disease which wiped out the balsam-fir sawfly..." (Cumming, 1953).

Now, as descriptive as such statements just quoted may be, they should not be interpreted as suggesting that a given species of insect can be eradicated from a given area by a disease agent. If the area is very small, it is conceivable that all of the host insects in that area could be eliminated by disease just as they could with chemical insecticides. However, for an area of any size, eradication by disease (occurring naturally or induced artificially) is not likely if at all possible. The survival of the species is not, perhaps, so much the result of a true immunity of certain individual insects (as suggested by Glaser, 1915, in the case of a polyhedrosis of the gypsy-moth caterpillar) as it is a matter of chance in some of the insects being fortunate enough to escape contacting an infectious dose of the pathogen. As host numbers fall, their degree of isolation increases which, in turn, decreases the likelihood of contact with the infectious agent. Furthermore, small areas in which the microorganism does kill all members of a species are quickly invaded by healthy individuals and, in most instances, the population density eventually is largely restored.

The relatively few epizootiological studies in which morbidity and mortality figures were actually recorded, indicate that disease can so reduce the number of insects in a given area that this state of affairs may, with a fair degree of accuracy, be recorded as a 98 or 99 per cent morbidity or

mortality. For example, Dirks (1943) recorded mortalities from disease (polyhedrosis) as high as 97 and 98 per cent of the population (European spruce sawfly). Similarly, and with the same disease and insect, Balch and Bird (1944) calculated and reported mortality percentages as high as 99.3 and 99.7 per cent. These same authors record that in some situations no second-generation larvae could be found and therefore assumed the occurrence of a 100 per cent mortality of the feeding population. Although, of course, insects died from causes other than disease, in this latter instance the 100 per cent mortality had not previously occurred in the absence of the disease. Here, again, it is likely that the report of 100 per cent mortality was not meant to be taken as a literal absolutism. Rather, that for practical, as well as epizootiological purposes, one could proceed as though no living insects were present in the area concerned, since the sampling methods used throughout the investigation could not reveal any survivors, although the species had not in actuality been exterminated. That this was the intention of the authors is further indicated by a subsequent statement of Bird's (1948):

[The disease] does not completely eliminate the insect from an area as it tends to disappear from a stand while there are still a few uninfected larvae remaining.

Even in a single epizootic, what may appear to be a 100 per cent morbidity or mortality in a prescribed area may, as the result of newly hatched larvae coming into the area, in a few days revert to a lower percentage. Thus, in artificially induced epizootics of a polyhedrosis in the alfalfa caterpillar, Thompson and Steinhaus (1950) record 100 per cent morbidities on the fifth and sixth days of an epizootic, but only a 90 per cent morbidity on the seventh day. Characteristically, however, in most of the artificially induced epizootics reported by these authors, after an incubation period of about five days, 100 per cent of the larvae was infected for the remainder of the test time (usually about three or four days). Occasionally, as the result of one of these epizootics, no living caterpillars could be found in the relatively small area to which the virus was applied, and for all practical purposes the mortality may be considered as being 100 per cent. In all probability in an area the size of several acres, enough individuals would escape to reproduce the species. In this instance there is little value in such speculation since with the next crop of alfalfa, if not before, adults from neighboring or distant fields would come in to oviposit the eggs for the next generation.

Thus, it appears that naturally disseminated disease agents do not ordinarily annihilate an insect population. Nevertheless, disease-producing microorganisms, like parasites and predators, can eradicate populations in small areas. Normally, these areas will be recolonized by members of the species population from other areas. However, in most instances in which disease breaks out in a population, eradication is not complete—largely because of chance and rapid reinfestation. Areas in which the population has been eradicated may continue for varying lengths of time to harbor the pathogen through the latter's normal capacities for survival or through its ability to form a resistant stage; or the pathogen may die out altogether.

4. Is disease capable of bringing about prolonged or permanent (as distinguished from temporary) natural control?

Most field entomologists are of the opinion that disease epizootics are, like insecticides, of a catastrophic nature and of only temporary control value. In a sense they are largely correct; but there are exceptions and room for differences in interpretation, and the analogy between microbial control and insecticidal control is not always valid. However, before coming to any conclusions as to whether or not disease is capable of inducing permanent control, the answers to two subsidiary and closely related questions should be considered. These are: What takes place when a microorganism pathogenic for insects is introduced into a new area? Can a pathogen be "established" in an area where there are susceptible hosts?

The matter of the feasibility of "introducing" pathogens into an area where they are not present but where there are susceptible hosts, needs considerably more study. It may well be one of the most important and promising aspects of insect pathology from the standpoint of biological control. In the history of insect pathology, the word "introduce" has been used in referring to entomogenous microorganisms that have been introduced into small areas, such as orchards, and that have been brought from one country or continent and liberated into another; in either case the microorganisms are not known to exist in the area concerned. The effects of such introductions have varied from what has been termed "successful introductions" to failures. (It would probably be well in this connection if insect pathologists adopt the terminology used by entomologists generally: that is, the term "introduction" would be employed to mean the bringing of microorganisms into an area where they did not formerly exist; the term "colonization" might then be used to indicate the release of organisms in an area where they are already known to occur.)

During the early years of the present century, state authorities in Florida advocated the introduction (and colonization) of certain fungi into citrus orchards for the purpose of controlling whiteflies and scale insects. As appraised by Morrill and Back (1912), such introductions by artificial means were, in themselves, successful but reintroductions or colonizations did not appear ever to increase in their effectiveness against the insects after once becoming generally established in a grove. The uselessness, at times, of introducing entomogenous fungi into areas where the spores are already plentiful, that is, where the saturation point has already been reached, was highlighted by Billings and Glenn (1911) in their report on the white fungus disease of the chinch bug in Kansas. They found the fungus to be present in such great abundance in infested fields that any artificial introduction of spores would be too insignificant, by comparison, to be of practical use. Parenthetically, it might be pointed out, however, that one should not generalize too broadly from the conclusion of these authors since even though a pathogen is present in a field in great numbers, if it is not situated in a place where it can contact or be contacted by a susceptible host, there is little likelihood of an epizootic resulting. According to Speare and Colley (1912), Clinton concluded that *Entomophthora aulicae* Reich could be of practical value in fighting the browntail caterpillar if the fungus was dis-

tributed in the field before the natural disease could develop under ordinary weather conditions, thereby giving the introduced fungus time to develop more generations, and consequently infect more larvae than would occur in the later-starting natural epizootic. In California, the polyhedrosis virus affecting the alfalfa caterpillar may abound in the soil, surface debris and irrigation water, but the caterpillars feeding on the tops of the alfalfa plants remain out of contact with the virus that in time will be distributed to the plants by gusts of wind. Before the latter occurs, however, the insects may be infected simply by distributing the virus over the parts of the plants on which they are feeding. In other words, the abundance of a pathogen in the general area of the insect's infestation does not necessarily mean that the artificial distribution of more of it is of no value. Each situation has to be appraised separately and on its own merits.

The introduction of a pathogen into an area where it is absent but where a susceptible host is present may, as in the case of the milky diseases of the Japanese beetle, be effective in establishing it and making it an effective and "permanent" agency of control. On the other hand, the introduction may be successful in establishing the agent but only in the sense that from time to time thereafter the pathogen may be found even though it does not constitute a continuous, prolonged, or "permanent" control of the host insect. This is probably one of the easiest ways in which to interpret, for the time being, some of the introductions that have been made of insect viruses.

In Europe, introductions of virus material have been attempted from one forest to another, with varying results. Various methods have been used, one of the favorite being the collecting of forest litter from areas in which the disease had recently occurred and then the transferring of this material to uninfected areas (Růžicka, 1924; and others). More recently several introductions have been made in Canada, which apparently have been successful in establishing the diseases concerned. According to Balch (1946), dried extract of diseased European spruce sawfly larvae was used to establish a polyhedrosis of this insect in Newfoundland. Prior to its introduction no diseased larvae had been found on the island, but before long the disease was prevalent over considerable areas surrounding the points of liberation. In 1950, Bird told of introducing a virus disease of the European pine sawfly into southern Ontario from Europe. The disease was unknown in Canada. Bird (1952; 1953) found the sawfly larvae to be very susceptible to the disease, and effective control was obtained following the artificial distribution of the virus. However, Bird did not comment on the establishment of the virus from the standpoint of its permanency. In the case of the European spruce sawfly, Bird (1954) found that virus, sprayed on seven trees in a virus-free section, spread over an area with a radius of approximately 2,600 feet by the end of three generations of the insect about a year later.

Many entomologists have held the belief that a newly introduced disease, like some newly introduced parasites or predators, is most effective from the standpoint of control at the time of its introduction and for a short time thereafter. This was early advocated in the fungus infections. For example, Clinton, 1908 (Speare and Colley, 1912), concluded that the browntail fungus would be of practical value against the browntail cater-

pillar if the fungus were introduced into territory in which the natural disease was not in evidence. It does seem to be true that although many years may elapse following the accidental introduction of an insect pest before any diseases affecting it appear, when the malady does strike it is with an apparent suddenness and thoroughness which may later subside to the more usual type of epizootiological fluctuations. There was no evidence of disease among chinch bugs in this country for about eighty years following the recognition of the insect as a serious pest. When the so-called "white fungus" appeared (about one hundred years after the insect's appearance) it seemed to spring up abundantly and simultaneously in widely separated localities. The gypsy moth was brought to the United States from France in 1869, but there is no record of its well-known polyhedrosis prior to 1900. By 1913, when Glaser (1915) made a survey of the region, the disease was flourishing throughout the entire territory infested by the insect. The suddenness with which disease may appear in insect populations has been frequently reported; the dynamics of the epizootics that occur in these instances as compared with those that occur more regularly or consistently require more concentrated investigation.

The apparent catastrophic rapidity and thoroughness with which natural epizootics may strike a population frequently hide the fact that the pathogen remains in the area ready to attack again when enabled to do so by an adequate population density and other factors. One occasionally hears it said that diseases strike rapidly and then leave the scene. While this is often the case, the point being made here is that the apparent "leaving the scene" is frequently simply a manifestation of the state of affairs that must exist when a population is drastically reduced by the disease. Actually the pathogen, perhaps in a resting or latent stage, may remain at the scene, but unlike, for example, parasites and predators which may be plainly visible, its presence remains undetected until it can show itself in the form of diseased insects. Thus, spores of certain entomogenous fungi may be continuously present in large numbers in fields ready to attack susceptible insect hosts, but these spores may remain inactive until appropriate conditions of temperature and humidity prevail. Similarly, certain viruses may remain virulent though inactive in fields or forests until the susceptible host insect increases in numbers to a point that an epizootic results. Insecticides, on the other hand, are usually dissipated in a relatively short time and do not remain in field crop, orchard, or forest between outbreaks of the insect.

In artificially induced epizootics for the purpose of suppressing or controlling a given population of insects, the residual or dormant pathogens in the general area may be of minor consequence. Frequently, the natural epizootics are delayed until the insect has already caused considerable damage. Artificially applying the pathogen earlier than it would be redistributed by nature is analogous to the use of insecticides as far as the effect of the application is concerned. Indeed, preparations of entomogenous microorganisms applied as sprays or dusts are sometimes referred to as "living insecticides." For instance, the polyhedrosis virus affecting the alfalfa caterpillar is plentiful in the soil, surface debris, and irrigation water in alfalfa fields. Not until late in the growing season are virus and caterpillar brought

together to produce epizootics. In the meantime, lethal concentrations of the virus may be applied directly to the leaves of the plant where the actively feeding caterpillars ingest the virus and succumb to the disease it causes.

The type of "permanent" control exhibited by successfully introduced and well-established parasites and predators has so far been definitely accomplished with microorganisms in only one well-known instance. We refer to the introduction of the bacteria causing milky diseases of the Japanese beetle. Ordinarily, little is accomplished by attempting to "establish" a microbial pathogen in the sense that this is done with insect parasites and predators. In the milky diseases of the Japanese beetle, however, this has been accomplished. Spores of the bacteria causing these diseases may be distributed in areas infested by Japanese beetle grubs with the result that the grub population is gradually reduced to a nondestructive level. Of interest to us here is the fact that once the turf or soil is impregnated with the spores of the bacilli, it remains in this state indefinitely or for long periods of time. When healthy grubs ingest soil containing the spores, they are infected, eventually die, and their disintegrating bodies liberate still more of the spores into the soil. This "permanency" of the pathogens may result from naturally or artificially introduced spores.

Perhaps obvious, but nevertheless significant, is the fact that prolonged and permanent suppression of an insect population by disease frequently occurs in confined populations, such as those held in rearing jars or cages. When the pathogen is particularly virulent, the population may be completely destroyed. On the other hand, chronic or low-grade infections may persist in a population, serving the while as a check on the number of insects making up the population. Thus, when *Tribolium castaneum* Herbst. populations suffer infection with the protozoan *Adelina*, the level of population density may drop as much as two and a half times and maintain itself for a significant period of time (Park, 1948).

Among those who have stressed the temporary nature of disease as a mortality factor are Ulyett and Schonken (1940). These authors, working with a fungus disease of *Plutella maculipennis* Curt. emphasize their conclusion that the "appearance of this disease in the field is sporadic and it cannot be regarded as a permanent member of the somewhat extensive complex of parasites associated with *Plutella*." And further, "Except in special areas, fungi can only afford a temporary remedy comparable to that afforded by insecticides." To the student in insect pathology it is difficult to relate these statements to certain others, for example:

On the basis of his studies on the *Empusa* disease of the green apple bug in Nova Scotia, Dustan (1924a) avers that since 1917 the insect has been gradually decreasing in numbers; that at the time he writes it appeared certain "... that the disappearance of the Green Apple Bug was due wholly to the work of this fungal parasite." Similarly, Speare (1922), in explaining the relative unimportance of the citrus mealybug in Florida, states that "It is reasonable to believe, owing to the wide distribution of the fungus, that similar epidemics were present elsewhere, and that as a matter of fact such widespread destruction of the mealybug has been going on for years, ..."

The work and conclusions of Ulyett and Schonken (1940) justify consideration in some detail. The following is an abbreviated account of their principal findings as these data are related to the matter we are discussing:

The normal population of *Plutella maculipennis* Curt. exhibits fluctuations about an average density typical of a population in a state of equilibrium. As shown in figure 1, line AA¹ indicates the average density of such a population. In this situation the insect causes no damage to the crop at the peak periods and is considered to be under the control of its natural enemies (parasites and predators). Because of unfavorable weather

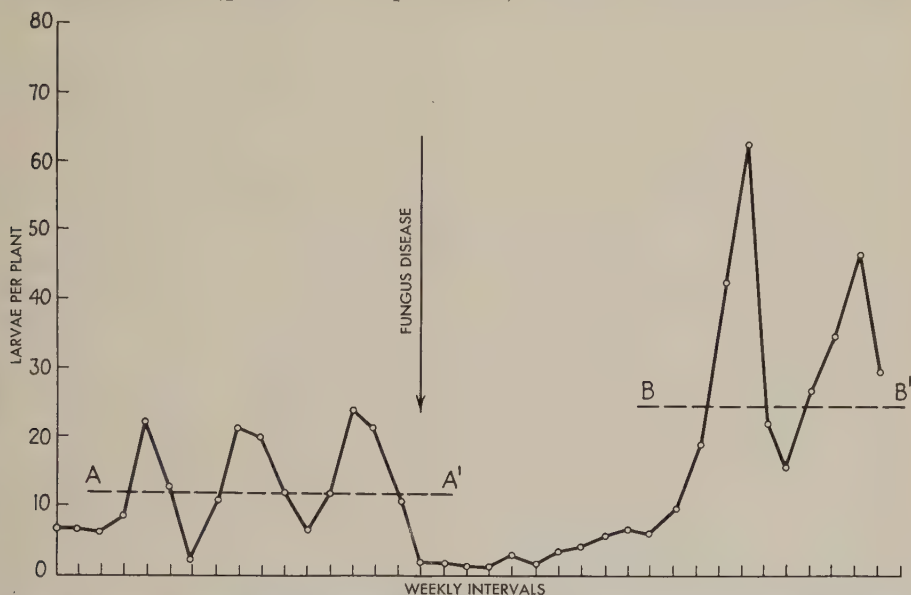


Fig. 1. Curve showing the progress of a population of *Plutella maculipennis* Curt., with an intervening epizootic caused by *Entomophthora sphaerosperma* Fres., as studied by Ulyett and Schonken (1940).

conditions, no cases of fungus (*Entomophthora sphaerosperma* Fres.) disease were found among the larvae during this period until the sudden intervening appearance of disease at the point indicated. The attack by the disease was followed by a rapid decline in the number of host individuals, and a low population was maintained throughout the ensuing period of favorable weather. With the disappearance of the disease, the host population quickly recovered. Since the parasites and predators ordinarily responsible for control had been largely destroyed during the epizootic, the population was able to build itself up to much greater proportions than had previously been the case. A new series of fluctuations then became evident, and these were taking place about a new average density represented by the line BB¹. This was approximately twice the value of the previous mean AA¹, and economic damage to the crop occurred at or near the peak periods.

From these observations, Ulyett and Schonken (1940) conclude that the epizootic had increased the average density of the host population, at least for some time, and that:

...the initial, sweeping reduction of the hosts has proved to be ultimately disadvantageous from an economic point of view. From a study of the survival values, the indi-

cations are that the change may be a permanent one which will survive until such time as another factor, having the reverse influence on the population trend, is introduced into the environment. To establish this in the field, however, will require continuous records extending over a long period.

Pertinent to the matter here being considered is their explanation of the phenomenon:

The intervention of the disease in an existing equilibrium system resulted in the replacement, by destruction, of permanent mortality factors by a temporary mortality factor (the fungus). When the latter disappeared, the host population was able to recover more rapidly than its parasites and thus to swing to a higher density level than before.

After again pointing to the disease as a temporary mortality factor, they close their paper with this rather provocative statement:

They [fungi] cannot, therefore, normally be included in the material at the disposal of the worker in this field [biological control].

It is probable that Ulyett and Schonken did not intend that this last statement should be accepted as a generalization pertaining to all fungus diseases of insects. Certainly there is ample evidence that entomogenous fungi can suppress insect populations without there being a subsequent sudden rise in the population to new levels of average density. Nor should the pessimistic tone of the statement as it pertains to biological control be allowed to discourage continued efforts to use these organisms in the control of insect pests in situations where benefit can be obtained.

That the effect of epizootics of the type described by Ulyett and Schonken can be of a temporary nature, there is no doubt. It is important, however, that we keep in mind just what is meant by the use of the word "temporary" in these situations. As was pointed out a few paragraphs back, a fungus or its spores may be permanent as far as their presence in an area is concerned but temporary (that is, sporadic) in their activities or effectiveness. Since this latter characteristic depends largely on suitable weather conditions, we might say that the "temporary" quality of their action is a function of the weather. In areas where the conditions of temperature and humidity are appropriate over long periods of time, the fungi are usually effective over the same long periods of time (for example, see Speare, 1922; Dustan, 1924*b*; Pickles, 1933). On the other hand, when the appropriate conditions of temperature and humidity are spasmodic, the activities of the fungus are likely to be spasmodic; and when the duration of the appropriate conditions of temperature and humidity is what might be called "temporary," it would appear justifiable to designate the disease that coincides with this duration as a "temporary mortality factor." Ulyett and Schonken apparently did not speculate on the possibility of frequent attacks of the disease, favored by appropriate weather conditions, being able to keep the population at low levels for a significant part of the time. However, in their comments likening the disease to an insecticide in its action they imply that repeated attacks (equivalent to repeated applications of insecticides) may "permit the pest to become even more abundant than formerly."

From what we have said in the preceding paragraphs, it would appear that in answering the question as to the permanency of natural microbial control, the following points might summarize the matter:

Under natural conditions it may be assumed that a particular pathogen is more or less continuously present in an area where it gives rise to epizootics of disease in populations of its host. Of course, the removal of or changes in the appropriate microenvironments will affect the constancy of their presence, as will the manner in which the area is "cleaned up" after an epizootic. The latter phenomenon undoubtedly comes about through such mechanisms as the washings of rain, the mortality of the pathogen, new growth on the food plant, and the like. However, the pathogen has to invade or get into the insect population as well as be in the general area. How this is effected will be influenced by the nature of the micro-environment, the habits and behavior of the host, physical conditions, population density, et cetera. It follows that when conditions (population density and physical conditions) are right the disease will become apparent in the population.

The correct level of population density would appear to be very critical for the inception of natural epizootics; in most instances the density has to be fairly high. If the "economic level" of the host density is considerably lower than the "threshold level" of the disease, then the control will be temporary and act like chemical-control measures. If the economic level of the host density is higher than the threshold level of the disease, then the control can be just as permanent as any other form of biotic control. The threshold of the disease will be modified (at least in certain entomogenous fungi) by the physical factors of the environment. Thus in the example (*Plutella*) furnished by Ullyett and Schonken, under one set of conditions (adequate moisture, et cetera) the threshold was below the economic level, while under other conditions the threshold was above, or near, the economic level.

From this it seems reasonable to conclude that disease will ordinarily give only temporary control and, from the practical standpoint, we must, in these cases, devise means of lowering the thresholds (as has been accomplished with the alfalfa caterpillar, for example, by spraying virus on infested crops). In some instances, however, pathogenic microorganisms may give prolonged or "permanent" control (as in the milky disease of the Japanese beetle); but these cases appear to be exceptional and in the minority on the basis of data now available.

5. What effect does disease have on other density-dependent control factors, such as insect parasites and predators?

The observations of Ullyett and Schonken (1940), referred to in the last section, highlight the need for close attention to the interrelations between microbial pathogens on the one hand and parasites and predators on the other, when the activities of these two groups of biotic agents overlap or otherwise come in contact. As pointed out by these authors, disease agents may adversely affect parasites and predators in two principal ways: (1) *directly* by infecting the parasite as well as the host (sometimes possibly infecting the immature stages of the parasite developing within the body of the host), and, less directly, by so depleting the host insect of available food materials required by the parasite that the latter dies of starvation; and (2) *indirectly* by reducing the host population to a point where the adult parasites or predators are unable to find enough hosts on which to

breed or on which to feed. We shall here be concerned with both of these effects, although the emphasis will be on the latter.

Although entirely germane to our subject, we shall not repeat here the evidence presented by Ulyett and Schonken to support their conclusion that the fungus concerned, while it produced a marked immediate reduction in the host (*Plutella*) population, was ultimately responsible for an increase in the average density of the host. Furthermore, the disease replaced or effected the destruction of the regular parasites and predators which, when the disease had run its course, were unable to recover as rapidly as the host population which established itself at a higher density level than before.

From the theoretical standpoint, Ulyett and Schonken (and Ulyett, 1953) analyzed their findings on the assumption that since the fungus disease appeared to be entirely dependent on the weather, it may be classed as a density-independent mortality factor. Therefore, according to these authors, since it cannot influence the average population of its host directly, any such effect must be produced indirectly through its reaction on the population of natural enemies. (Here again, however, we must interject the thought that although the fungus disease is dependent upon the weather, this does not make it a density-independent mortality factor. In reality, all mortality factors are dependent upon the weather; obviously no factor would be density-dependent in an unfavorable environment.)

Finding support in Volterra's (1931) "Law of the Disturbances of the Averages," and certain conclusions of Nicholson's and Bailey's (1935), Ulyett and Schonken maintain that, in the majority of cases, the disease (comparable with Nicholson's and Bailey's "destructive environmental factor") will result either in the final density of the host population becoming stabilized at a higher level than before, or in its remaining unchanged; only rarely will a decrease in the population level occur. In this conclusion, the South African authors liken the mode of action of the fungus to that of an insecticide, conforming to the factor causing the destruction of species in Volterra's formulae. They venture no opinion on the feasibility of artificially reintroducing the parasites and predators when the host population begins to rise again after the disappearance of the disease. Such a concerted procedure is certainly not beyond the range of practicality in some instances. In fact, in reporting on the control effects of a polyhedrosis of the European spruce sawfly, Bird (1948) states that the activities of introduced parasites limit the rate of population increase when the disease is absent or at a low level. Whether such procedures would work in cases involving weather-dependent fungus epizootics remains to be seen.

As we have warned earlier, the idea that certain fungus diseases are density-independent mortality factors cannot be accepted in the literal sense, and, certainly, it does not permit wide generalizations covering other types of diseases affecting insect populations. There are known situations in which there is no great interference between the disease and the parasites of the same insect host, or in which the parasites or predators are able to build up rapidly between epizootics constituting an insurance against reduction in the effectiveness of disease, or in which the disease does not reduce the population to a level inimical with the perpetuation of the parasite (for example, see Balch, 1946a).

In other instances, as in the case of the polyhedrosis of the alfalfa caterpillar, the parasites from adjacent fields or surrounding territory rapidly penetrate anew the area in which the epizootic has occurred. Michelbacher and Smith (1943) point out that when the disease occurs in epizootics of great intensity many of the parasites (*Apanteles medicaginis* Mues., which parasitizes the first three instars of the caterpillar) are destroyed along with their hosts. On the other hand, in epizootics of moderate intensity, the majority of the caterpillars killed by the disease are in the last two instars. The insect parasites may kill the smaller larvae while the virus operates against the larger larvae. Thus, the virus may indirectly be responsible for a more dense population (relative to the caterpillar) of *Apanteles* in adjacent fields than would otherwise be present. Further study has indicated that under natural conditions there is no great direct conflict between virus and parasite. Thompson (1951) recommends, however, that in artificially applying the virus for control purposes, it is advantageous, when possible, to withhold application of the virus until the caterpillar population is in the second and third instars so that the degree of parasitization can be determined. If parasitization is high, no further treatment may be necessary. Thompson (1954, unpublished) has found that, when correctly timed, the virus treatment greatly reduces the caterpillar population without having a marked effect on the parasite. Parasite larvae can develop normally in a virus-diseased caterpillar providing the host does not die before the parasite has completed its development. In nature, and in controlled field treatments, the parasite larvae usually emerge and pupate before the infected hosts die. Furthermore, Thompson feels that data presently available indicate that virus treatment of economic populations of the alfalfa caterpillar increases the chances of the *Apanteles* parasite in controlling the following generation of caterpillars.

European observers of a polyhedrosis (*Wipfelkrankheit*) of the nun moth caterpillar, have noticed that in areas where tachinids parasitize the insect, usually it is the older larvae that are parasitized; but where the polyhedrosis also occurs, heavily diseased fifth-instar larvae are not parasitized (Niklas, 1939). The high incidence of virus disease in heavily infested areas causes many of the tachinids to migrate to the less infested areas. Gösswald (1934) observed that the parasite *Sarcophaga shützei* Kram. would not parasitize healthy larvae of the nun moth or the gypsy moth, but it did attack individuals suffering from polyhedrosis virus diseases. On the other hand, Masera (1948) reports just the opposite in a protozoan disease in that healthy larvae of the European cabbage butterfly were parasitized by *Apanteles*, whereas those infected with a species of Microsporidia were not.

As noted by early workers in biological control, the presence of disease in a host population may at times make the introduction and establishment of insect parasites and predators difficult. Howard and Fiske (1911), for example, found a polyhedrosis of the gypsy moth caterpillar to interfere seriously with the work of colonizing *Anastatus* and *Calosoma*.

Under some circumstances, the presence of a disease apparently may cause an actual increase in the proportion of parasites to the host insects. For example, King and Atkinson (1928) during their studies on the biologi-

cal control of the red-backed cutworm, *Euxoa ochrogaster* (Guen.), in Saskatchewan found this to be true. As shown in figure 2, in one area studied, the mortality from disease rose from 13 per cent on May 17, to 73 per cent on June 25. During the same period, the moth emergence fell from 69.8 per cent to 1 per cent. When a comparison is made of the parasitism (by *Meteorus vulgaris* Cress. and other species) curve with the other two, an

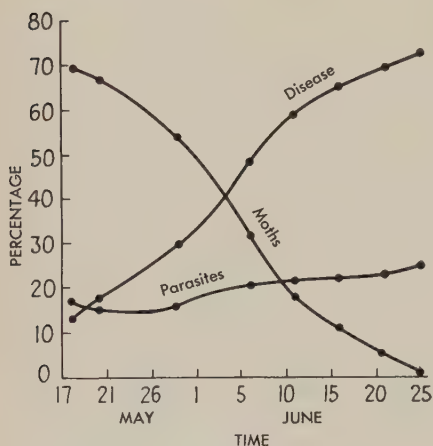


Fig. 2. Graphic representation of the mortality of *Euxoa ochrogaster* (Guen.) from insect parasitism and disease, as studied in Saskatoon, Canada, by King and Atkinson (1928).

important fact is revealed. During the period of June 15 to 25, the percentage of moth emergence decreased from 13 to 1. Therefore, King and Atkinson conclude that the disease was not only effective in destroying most of the larvae present during the year 1925, but also was effective in causing a greatly increased proportion of parasites to moths emerging, thus favoring the possibility of high parasitism the following year.

There is another disease-parasite phenomenon worthy of note. In 1911, Howard and Fiske hypothesized that when an insect pest is ordinarily controlled by parasites, "it is probable that a long time will elapse before it will again encounter the combination of favorable circumstances which make possible abnormal increase," which, in turn, would enhance the possibility

of an outbreak of disease. These authors go on to say that when adequate control by parasites is lacking, reduction in numbers through disease is not likely to result in more than temporary relief. They tell of localities where, in their opinion, the destruction of a great majority of gypsy moth caterpillars by what is now known to be a virus disease was the only thing that prevented the insect's extermination in those localities. They reach this conclusion on the assumption that had the disease not intervened the entire supply of available food would have been consumed before the insects had completed their development, and the insects would all have starved. Thus they believed that the gypsy moth would have been much less abundant generally had it not been for the prevalence of disease, and that, as far as natural enemies were concerned, the best control could be had with parasites and predators. They further concluded that since the virus disease in itself did not naturally control the gypsy moth in its native country (Russia), "then something better than disease must be found to control it in America." This deduction increased their determination to introduce appropriate parasites and predators. In this connection it is interesting to note that Komárek (1950), as the result of his studies on certain forest Lepidoptera and Hymenoptera in Czechoslovakia, concluded that "parasites are never able to control their host so as to prevent it from multiplying from time to time

in mass in its original home. By themselves the parasites cannot stop an eruption once started. The mass multiplication is terminated in most harmful insects always by the epidemic spread of a virus infection . . ."

To some extent, Howard and Fiske were justified in their discouragement over being unable to rely on disease as an adequate regulatory factor. Nevertheless, today we would find some of their conclusions to be rather tightly drawn. As did many workers after them, Howard and Fiske expected too much of disease as a regulatory or consistent control factor unaided by the help of man. Because microbial agents were one of the groups of living organisms comprising what is known as the "natural enemies" of insects, and since other natural enemies such as parasites and predators were introduced and handled in a manner intending that they become established as part of the natural fauna of the region, it is not surprising that many of the earlier workers expected disease to perform in much the same manner as parasites and predators.

Disease agents, or infectious materials of one kind or another, were frequently brought into a new area and "liberated" in a manner similar to that used in introducing beneficial insects. It was a long time before general recognition was given to the idea of distributing microbial agents in the same manner that insecticides are applied. This is not to say that introductions, liberations, or "plantings" of entomogenous microorganisms are not feasible in some situations. They are, as we have explained some pages back. Nevertheless, it is probably true that had Howard and Fiske had available modern methods of applying sprays and dusts, their conclusions and implied generalizations might not have been put quite so dogmatically. Furthermore, the over-all effects of disease that these authors disparage need reexamination in view of contrary data that have been forthcoming in recent studies of other forest defoliators. As an example, the thesis that in a given locality "complete extinction" by starvation would occur were it not for the intervention of disease probably requires strong qualification. Indeed, it is known that sometimes the sequence is reversed. The parasites and predators of a pest may be destroyed through the use of insecticides, allowing the host insect to increase at such a rate that the parasites and predators exceed their food supply and starve before disease has had a chance to enter the picture. In other cases, to be sure, insecticides may kill off the parasites and predators, permitting the host insect to increase in numbers to a density that promotes the outbreak of disease.

In instances in which the host insect is subject to the attack of both parasites (or predators) and microbial pathogens, it cannot reliably be predetermined which of the two biotic forces would be of greater importance from the standpoint of the regulation and/or control of the population. From a regulatory standpoint we should be inclined to agree with H. S. Smith (1935) that entomophagous insects would, in general, have to be ranked as the most important, "although in certain specific cases infectious and contagious diseases would precede them." Bucher (1953), for example, in a study of the extent of mortality suffered by a budworm attacking fir in Europe, concluded that although both larvae and pupae of the budworm were attacked by a complex of insect parasites, these were of secondary

importance to a virus disease in reducing the population. From the standpoint of control, however, the comparative effectiveness of the two agencies depends not only upon the numerous factors and conditions involved in each instance, but upon knowledge and epizootiological information forthcoming in future studies. At the present time we consider it virtually impossible to make a valid appraisal of the over-all effectiveness of microorganisms as compared with entomophagous insects in the natural control of insects. Furthermore, as far as basic principles are concerned we can only make the broadest of comparisons, for example, that while predators usually breed more slowly than their prey (Haldane, 1953), pathogenic microorganisms multiply much more rapidly than their hosts.

It would appear that disease usually has a higher "threshold" of operation than do insect parasites in a given population. When present, parasites and predators will normally be keeping the population levels below the thresholds of disease. In unusual instances (as with the fungus disease of *Plutella*), a change of conditions may bring on the disease at a low threshold and eliminate the parasites. When parasites and predators fail, or are absent, the populations may rise to a point where disease has a greater opportunity to act. This supposition, however, refers only to natural circumstances and does not apply to situations in which the pathogen is artificially applied.

We have discussed situations in which parasites and disease function in antagonism to each other, and others in which they work in concert, and we have seen that in some instances diseases may enhance the activity of parasites. It remains to be mentioned that there is one relationship between these two groups of organisms in which the behavior of the parasite aids the disease. This has to do with the natural transmission of insect pathogens from one host to another, and forms a logical part of our discussion of the next question.

6. What relation has the mode of transmission or dissemination of a pathogen (or its capacity to spread) to the epizootiology of disease in insect populations?

Earlier in this paper were listed three attributes that characterize microbial agents responsible for epizootics in insect populations. The third of these attributes concerned the capacity of a microorganism to spread. Under this designation was included the automobility of the pathogen, distribution by other living organisms, and mechanical transmission by such agencies as wind, rain, and the like. It will be our purpose here to elaborate this subject in order to have a clearer understanding of its relation to the epizootiology of diseases occurring in insect populations.

Many microorganisms pathogenic for insects are, at one time or another in their life histories, capable of considerable self-movement. This may be accomplished through organs of locomotion (flagella, cilia, pseudopodia, et cetera) or by such extraordinary devices as the violent discharge of conidia into the air by certain fungi.

It is difficult to evaluate the importance of such automobility as far as its relation to epizootic dynamics is concerned. In most instances the pathogen is not dependent solely on its own ability to move in order to find a new host. Nevertheless, the autolocomotion of certain stages of some pathogens

(for example, the zoospores of chytridiaceous and blastocladiaceous fungi, the amoebula of Microsporidia) is necessary to provide for the continual development of the organism. The pathogens of insects living in water may enhance their distribution through the host population by flagellar, ciliate, or pseudopodial locomotion. The same may occur in moist situations in terrestrial locations.

The ability of fungi of the order Entomophthorales to discharge conidia away from the host on which they are growing undoubtedly enhances the microorganism's ability to spread. The mechanism involved is essentially one in which, through absorption of water, there is an expansion of the conidium and the basidium of the conidiophore in which it is held. The resulting build-up of pressure soon is great enough to rupture the wall in a circle near the base of the conidium. As a result of this rupture the conidium is freed and discharged violently into the air for a distance of from several millimeters to as much as an inch or more, or still farther if caught by air currents.

When discharged, the relatively short-lived conidium, if it comes in contact with a suitable host, adheres to it, sends out a germinating hypha, which enters the body, and the host becomes infected. The capacity of the conidium or spore to adhere tenaciously to its new host is, in itself, a mechanism that increases the pathogen's "capacity to spread." To be sure, the ejected conidium usually does not land on a new host; but other potentialities remain. If it lands in water, it gives rise to one or more hyphae that may branch and grow until the protoplasm is spent. If it lands on a dry surface, the conidium proceeds to form secondary conidia which are discharged in the usual fashion. If still not successful in finding a new host, tertiary conidia may be formed, and so on until it has come in contact with a susceptible host. However, there are still other devices possessed by these fungi to aid them in eventually finding a host. Instead of secondary conidia, thick-walled spores that are not discharged may be formed. Or, within the body of the insect, special "resting spores" may be formed that are highly resistant to conditions that would ordinarily destroy the conidia. The fungus is here preserved until, upon the disintegration of its host's body and/or the occurrence of appropriate conditions of moisture and temperature, it may again have the opportunity to infect a fresh host.

We have recited some of the details of certain phases of the life cycle of the Entomophthorales because these fungi so well illustrate the numerous mechanisms, devices, and processes by which a pathogen may directly or indirectly enhance its capacity to spread and, by itself, increase its distribution. Obvious is the fact that the chances of any type of autolocomotion making it likely for an obligate pathogen to find a new host will depend greatly upon the density of the host population or its habits of gregariousness or colonization. Since such contacts are largely a matter of chance, the more dense the population the more probable the pathogen will contact a new host.

At times, the movements of the host insect may be of some importance in the spread of a disease, even though the pathogen itself is in a passive state. Healthy insects in touching their dead or diseased fellows may acquire

the pathogen in numbers sufficient to cause infection in them. (It is important to remember that dead insects serve as a source of infection frequently to a greater degree than do living diseased insects.) Or, the healthy insects may make contact with infectious material left by the insect. Similarly, scavenging and other nonsusceptible insects may by purely mechanical means carry or transport the infectious agent from one place to another. Actual evidence of this type of transmission was obtained by Bird (1953), who found that polyhedrosis virus of the European pine sawfly carefully smeared on the trunk of one tree in a formerly disease-free plantation apparently became disseminated about sufficiently to enlarge the area in which mortality of the sawfly larvae occurred.

The movements of the susceptible host are, of course, themselves of importance in the spread of infection. Active individual insects pass from one contact to another, and the greater the number of contacts, other things being equal, the greater the chances of infection. An experimental example of this has been recently provided by Clark (1954) who treated several colonies of young tent caterpillar larvae with a polyhedrosis virus, and then placed them on plants on which there were untreated insects. According to Clark, the subsequent death of the insects not treated directly indicated that, under the conditions of the test, the disease may spread fairly rapidly among the larvae of a single generation. From what has been learned from epidemic disease among humans (for example, see Stallybrass, 1931), we might assume that in insects the chances of a disease spreading is increased in proportion to: (1) the number of individuals in movement; (2) the degree of the insects' susceptibility to infection; (3) the susceptibility of the populations with which they come in contact during their movements and at their destination; (4) the efficacy of the means of transmission; and often in proportion to: (5) the speed of movement; and (6) the degree of overcrowding during movement.

The transmission of microorganisms from one generation of insects to the next in association with the egg, is a well-recognized phenomenon in vectors of microorganisms infecting man and animals, in microbial symbiosis in insects, and to a minor extent in insect vectors of plant diseases. This manner of transmission is generally spoken of as "transovarial transmission" and may be broadly considered as including transmissions occurring when the microorganism is merely attached or otherwise associated with the exterior surface of the egg, as well as those in which the microorganism is incorporated within the egg and thus automatically transported to the young of the next generation. However, it is at times important to clearly differentiate these two types of transovarial transmissions.

As far as the pathogens of insects are concerned, it is known that transovarial transmission of certain protozoa and viruses does occur, but there is little or no evidence that the same occurs with bacteria or fungi capable of causing disease in insects. Some have maintained that the coliform bacterium responsible for bacterial dysentery in grasshoppers passes from one generation to the next through the egg but this viewpoint is not generally accepted and experimental evidence is lacking. It is conceivable, however, that eggs laid by an infected female could be mechanically contaminated,

but the fate of these bacteria associated with the egg masses in the soil and subsequent to hatching would seem to be a precarious one; although, again, infection from such a source is conceivable.

A classical example of the transovarial transmission of an insect pathogen from one generation to the next was Pasteur's (1870) discovery that such occurs in pebrine of the silkworm. The pathogen is the protozoan *Nosema bombycis* Naegeli, a microsporidian. (A considerable number of microsporidia appear to be transmitted via the egg.) In fact, this manner of transmission constituted one of the principal means of spreading the disease among sericulture nurseries. It was largely by preventing the use of such contaminated eggs that Pasteur was able to offer a means of controlling this disease which threatened the silk industry of France and Italy.

Eggs have been freed of microsporidia by immersing them in hot water baths at temperatures that killed the protozoan but did not harm the egg. Such experiences, however, do not prove that in some cases, or at certain times, the pathogens may not be transported within the egg itself. There is some evidence to support this latter possibility.

Transovarial transmission of insect viruses has also been known for a long time. Such transmissions have been reported for both polyhedrosis and granulosis viruses. Examples of these are readily found recorded elsewhere (for example, Steinhaus, 1949; Roegner-Aust, 1950; Bergold, 1953). Unfortunately, in most of these instances, as in most cases of the transovarial transmission of protozoa, it has not been made clear as to whether the agents are transmitted on the exterior surfaces of the eggs or within the eggs. It has been amply demonstrated that eggs that would ordinarily yield diseased larvae can be freed of the virus by immersing in such disinfectants as formalin, trichloroacetic acid, and the like. The relation between the manner in which a virus causes an epizootic and its transmissibility via the egg is indicated by Bird (1954) in a note relating to his studies on the polyhedrosis of the European spruce sawfly. Population trends on individual trees showed that epizootics each year start from the infection of a very small percentage of the larval population. Since Bird found very little if any virus to remain on the trees over winter, because of the cleansing effect of rains, he concluded that the initial infection each year is due to transmission of virus via the eggs of infected adults. On the other hand, Clark (1954) obtained evidence that a polyhedrosis virus of the Great Basin tent caterpillar survived the winter on the host plant and was a potential source of infection for developing larvae. Clark found transovarial transmission to play an important role in carrying the virus through the 9- to 10-month period during which no susceptible stage of the insect is present.

The significance of the transovarial transmission of microorganisms pathogenic for the ovipositing species is not clear. No one has been able to estimate accurately the degree to which such transmission takes place in field populations. Undoubtedly, when it does occur it ensures infection of the succeeding generation in the event other means of transmission fail. Under ordinary circumstances, however, with other means of transmission functioning it is unknown what effect a significant amount of transovarial transmission would have on the population dynamics. One conceivable effect might be

that a considerably greater number of young or immature insects would be infected than might otherwise be the case.

So-called "true biological transmission," in which the microorganism actually multiplies or develops within the body of an insect vector is not known to occur in any of the diseases of insects. The nearest to this happening is in the case of some of the insects which acquire certain nematode (Nematomorpha) infections by swallowing other insects that serve as intermediate hosts. When the intermediate hosts (for example, Chironomidae, Ephemeridae, Trichoptera) are ingested by a carnivorous or omnivorous insect, the worms complete their development in the latter, or final, hosts. Most nematodes are transmitted from host to host orally along with contaminated food, or by direct penetration of the integument, or possibly during copulation.

Although true biological transmission via an insect vector is unknown among the diseases of insects, mechanical transmission of infectious agents from one host to another by means of other insects does occur. Ordinarily this may occur during the normal activities of the insect parasites and predators of the susceptible insect.

Just how significant a role parasites and predators may play in the transmission of entomophilic pathogens is, however, difficult to judge on the basis of the few observations that have been made in this regard. The best evidence that such a phenomenon occurs is apparent in the case of certain virus and protozoan diseases. Theoretically, any parasite that travels from host to host, inserting its ovipositor into the body cavity of the insect may serve as a potential vector of disease agents developing or present in the body of the insect. Although predators usually destroy their hosts in the process of feeding on them, it is entirely possible that in their search for hosts, predators may mechanically distribute infectious material over considerable areas, thus increasing the likelihood of the pathogen being contacted by the susceptible host. That such may occur is evidenced by the credence given the role that insects, birds, and possibly even skunks, moles, and mice play in the dispersion of the organisms causing milky disease of the Japanese beetle (White and Dutky, 1940).

In 1915, Glaser speculated on the possibility that the virus responsible for a polyhedrosis of the gypsy moth caterpillar may have been introduced from its original source in 1905 with the parasites imported to aid in the control of the moth. According to Glaser, one of the tachinid flies, *Compsilura concinnata* Meig., is especially well adapted to aid in the rapid dispersion of the disease. He ascribed the prodigious increase in the disease as probably the result of the activities of this and other parasites. A similar possibility was recognized (Steinhaus, 1948) with *Apanteles medicaginis* Mues. in the case of the polyhedrosis of the alfalfa caterpillar. Experimentally, Thompson and Steinhaus (1950) showed that adult *Apanteles* were capable of transmitting infective doses of virus from diseased to healthy insects presumably as the result of stinging healthy caterpillars with ovipositors that had previously been used to sting diseased larvae. In the experiments performed, a contaminated *Apanteles* appeared capable of transmitting virus to three successive hosts at hourly intervals. There is a possibility that some

transmission of virus may also take place via the contaminated external body surface of the parasite. Mechanical transmission by ants allowed to feed on virus-dead larvae was also shown to be possible.

Another instance of parasite transmission of an insect pathogen was reported by Paillot (1924; 1937). According to this author, certain microsporidian and bacterial diseases of the cabbage butterfly apparently may be transmitted by *Apanteles* parasites. Similarly, Chorine (1930) suggests the accidental transmission through the agency of insect parasites of a microsporidian he found infecting the larva of the small tortoise-shell butterfly. Payne (1933) has presented evidence that the hymenopterous parasite *Microbracon* can transmit a microsporidian (*Thelohania*) from one Mediterranean flour moth larva to the next. Metalnikov and Chorine (1926) showed experimentally that bacteria responsible for disease in wax moth larvae could be transmitted by the chalcid *Dibrachys cavus* Wlk. (= *D. boucheanus* Ratz.). (See also Toumanoff, 1950.) Undoubtedly similar examples exist with other protozoan, virus, and bacterial infections.

Cannibalism is believed to aid considerably in the transmission of some entomophilic pathogens, such as that causing bacterial septicemia in grasshoppers. The failure to recognize the fact that the "*acridiorum*" bacterium can be transmitted through the cannibalistic habits of certain grasshoppers has been cited as one of the probable reasons why the use of the bacteria failed in instances where it was tried against noncannibalistic grasshoppers. In other instances, too, it appears that carnivorous insects, and insectivorous animals, such as birds, probably spread infectious agents after feeding on dead or dying insects.

What is known concerning the role of such factors as wind, rain, and irrigation water in the dissemination of insect pathogens is known largely by inference. The literature of mycology and plant pathology contains numerous accounts pertaining to the extramural aerial dissemination of plant pathogens, but very little of the work in insect pathology has been concerned with this aspect of transmission. Students of aerobiology have long known that dust and biological particles of many kinds can be carried by wind and air currents for long distances. Microorganisms (fungi) in the atmosphere have been recorded at altitudes of 36,000 feet; bacteria, as well as fungus spores, have been found at around 20,000 feet; yeasts and pollen grains above 16,000 feet; algae and moss protonema at 6,600 feet. Dust and microorganisms of many types are regularly found in abundance at the lower levels. This is true over the sea and in arctic regions as well as above land surfaces. Thus there is no question but that "the atmosphere is continually circulating large amounts of viable and nonviable material and that certain meteorological factors operate in the introduction and transport of these particles" (Proctor and Parker, 1942).

Since most microbial pathogens of insects at one time or another become integrated or associated with the soil or surface debris, it is reasonable to assume that they are commonly circulated through the atmosphere freely or along with dust particles. At least since the early observations by Reiff (1911), the wind has been thought to play an important role in disseminating pathogens from "infected areas" to "noninfected areas," from one field

to the next, and from one part of an orchard to another part. It is probable that in reality wind distribution of the pathogens occurs over a wide area and for long distances, at least up to several miles. Air currents probably are also responsible for widespread distribution except when hindered by winds, excessive rains, and natural barriers such as mountains, large bodies of water, deserts, and the like.

That limited local transmission of some insect pathogens may occur without being carried on dust particles or in the form of dust is indicated by experiments with the polyhedrosis virus affecting the alfalfa caterpillar (Thompson and Steinhaus, 1950). Under experimental conditions, the cadavers of freshly dead larvae, being fragile and liquefied, had a tendency to splatter under the influence of artificially created air flow (four miles per hour) and the whipping action of the alfalfa. Under such circumstances, clean alfalfa a foot or so away from the dead insects became contaminated. Once the cadavers had dried or hardened, no aerial transmission was detected. However, when infectious material was mixed with soil and allowed to dry, the directing of an air current across the soil onto clean alfalfa six feet away caused the plants to become infectious for the alfalfa caterpillar. Under natural conditions and under the influence of strong winds and air currents, it is to be expected that many of the microorganisms affecting insects would be carried along with dust particles for long distances (that is, many miles).

The dissemination of microorganisms along with dust particles is enhanced during dust storms or at times of unusually high winds. In addition to transporting the microorganisms, the dust particles may act as condensation nuclei for water vapor in the atmosphere and thus provide conditions of moisture and temperature favorable for the viability of the microorganisms (Proctor and Parker, 1942). Of course, microorganisms and their spores differ a great deal in their ability to resist the effects of desiccation and rays of the sun, so it is difficult to generalize on the extent to which these living agents are able to be transported through the air without losing their viability.

In this connection, it should be remembered that insects themselves may be passively transported by wind (for example, see Glick, 1942). Such insects may be in a diseased condition, or they may be dead of disease, and thus serve to carry pathogens from one locality to another. Such transportation may or may not be related to the migration or emigration of insects. Migratory movements undoubtedly aid in the distribution of microbial pathogens. The same statement probably applies to all types of insect dispersion. The range of distances involved is, of course, tremendous. As pointed out by Wolfenbarger (1946), it is in terms of inches for insects such as primary screwworm larvae and European corn borer larvae, and in hundreds of miles for migratory grasshoppers and sugar beet leafhoppers.

It has been shown that there is at least limited local dissemination of microorganisms in driving rainstorms. In fact, when considered in aggregate, the extent of displacement and distribution of microorganisms resulting from rain splashes or droplet splatter may be considerable. Well known is the fact that natural precipitation itself may contain microorganisms.

Rain water collected aseptically frequently contains microorganisms gathered from the air or from dust particles about which rain droplets have formed. Bacteria have been reported in rain and snow presumably far removed from their place of origin (Zobell, 1942).

Frequently overlooked is the role of irrigation water in the dissemination and redistribution of microbial pathogens of insects. Both the pathogen itself and the insects dead of disease may be carried considerable distances by run-off water or by the supply water if this is contaminated enroute to agricultural fields. At any rate, there is evidence that irrigation water may be important in the dissemination of entomogenous microorganisms within any one field, and pathogens may, by means of the water, be elevated to positions on growing crop plants where, after the water recedes, insects may ingest them while feeding on the plant.

When microorganisms are distributed by artificial means for the purpose of controlling an insect pest, additional factors must be taken into account. These include: (1) method of distribution (spraying, dusting, spot releases, et cetera); (2) form in which they are distributed (sprays, dusts, aerosols, et cetera); (3) quality of preparation (for example, viability of the pathogen, droplet size, et cetera); and (4) efficiency of application (that is, the human factor).

In general, microbial agents are relatively passive and have low degrees of mobility compared with parasites and predators with their "searching" power. This is probably one reason for the relatively high threshold exhibited by disease. It should also be remembered that, although microorganisms are living agents, in most instances their artificial dispersion has to be accomplished in a manner similar to that used with chemical insecticides. To be sure, a greater or lesser amount of spread of infection from diseased to healthy insect occurs as the result of contagion. Once an infection has taken hold in a population, there may or may not be a varying degree of dissemination by natural processes. Also, unlike chemical poisons (of which the insect must receive or gather a lethal dose) pathogenic microorganisms, once within a susceptible host, may of their own accord increase in numbers to a point capable of causing its death; although here, too, a minimum dose is required to initiate the infection.

In the case of chemical insecticides, the maximum economy of the poison may require an optimum droplet diameter for any particular insect size. It is also necessary to distribute the insecticide preparation as evenly as possible. In using nonvolatile insecticides against aerial or terrestrial insects, the droplets or particles of poisonous material should be so numerous that the insect will not fail to contact a lethal dose, but to attain this large number the droplets should not be so small as to drift past or around the insect in its flow streamlines. When spraying vegetation for insects, if the droplets are too small they do not hold enough material to impinge upon it, and evaporation is more likely to occur; also, since the smallest droplets tend to remain air borne longer than larger drops, they are more likely to drift away from the target area on wind and convection currents.

In summarizing his studies on the relation of particle size of insecticides to their application, distribution, and deposit, Potts (1946) points out that

fine atomization is necessary to obtain adequate distribution with low gallonage, but the droplets must be large enough to deposit on foliage and insects. The optimum droplet size for ground application apparently should be 20 to 80 microns in diameter. Droplets having a smaller diameter than 30 microns, as well as individual particles of very small size, are repelled by a field of resistance that surrounds all objects, including plants and insects. It should be remembered that wetting agents in aqueous sprays reduce the droplet size 30 to 50 per cent. For aerial application, if the areas are large and flat, the droplets should be 70 to 100 microns in diameter. In the case of certain forest insects on rugged terrain, the droplet size should be as large as 100 to 300 microns. According to Potts, other factors affecting particle or droplet size include: type of distributing device; concentration, density and type of insecticide; rate of volatilization of the ingredients; distance to which the particles must be drifted; wind and other meteorological factors; volume and compactness of foliage; and settling rate.

In the case of microbial control agents, the effective droplet size may, to some extent, be limited by the size of the microorganism. That is, the smallest effective size could not be smaller than the size of the microorganism. Fortunately most microorganisms (or their spores or resistant forms) do not exceed the 30 to 80 micron size recommended by Potts for ground application. In most instances, the desirable droplet size would be such as to encompass several individual microorganisms.

SUMMARY

Inasmuch as this paper is in itself a summarization of certain leading aspects of the effects of disease on insect populations, no effort will be made here to present a summary of the numerous points discussed. Instead, an attempt will be made to present only a brief resume of the principal concepts the author has reviewed and formulated.

Throughout the paper, disease dynamics and the role of disease in the natural control of insect populations have been emphasized. The epizootic wave is treated from the standpoint of insect populations, and the qualities of the infectious agent and the host insect, as they relate to the epizootic wave, are considered.

As a factor in the natural control of insect populations, disease may be characterized according to a number of general ecological principles. On the basis of present knowledge, the chief of these may be stated as follows:

1. In general, disease-producing microorganisms are density-dependent mortality factors. Large numbers of a susceptible host population, in the presence of an adequately disseminated infectious agent, are conducive to the outbreak of disease. However, disease may also manifest itself at low levels of the host population, and, under certain conditions, may even be effective as a control factor at low levels. An epizootic that breaks out when the density of the host population is high may gain a momentum that extends it for long distances regardless of the density of the population. When the infectious agent is artificially distributed, epizootics can be initiated in populations of low density. Epizootics of disease can be primarily responsible

for the cessation of the density increase and the "crash" of an insect population. It can be assumed that density-dependent and density-independent factors are both involved in the totality of epizootic dynamics, but there is little reason to question the generalization that disease is essentially a density-dependent mortality factor.

2. Weather conditions influence disease epizootics indirectly, primarily through their effects on the host insect. In general, conditions of temperature and humidity that are favorable for the insect are favorable for the development of the disease, and conditions unfavorable for the insect are usually unfavorable for the disease. Thus, the rate at which a disease spreads may be accelerated or attenuated depending on weather conditions. In the case of certain fungus diseases, but not most bacterial, virus, and protozoan diseases, adequate temperature and moisture may be required to initiate an epizootic since otherwise the conidia and spores of these microorganisms will not germinate or infect their insect hosts. However, although the development of fungus diseases may be dependent upon the weather, this does not make them density-independent mortality factors. In reality, all mortality factors are dependent upon the weather; obviously no factor would be density-dependent in an unfavorable environment.

3. Ordinarily, naturally disseminated disease does not annihilate or completely exterminate an insect species over any general area of the insect's geographic distribution. If the area is very small, eradication may be possible to the degree it might be if chemical insecticides were used. Nevertheless, populations of some insects can frequently be reduced 95 to 99 per cent through the natural occurrence of disease as well as by the artificial distribution of the infectious agent.

4. Although microorganisms are living agents, they do not function in all respects as do other agents of biological control, such as parasites and predators. The "staying capacity" of microbial agents may be transitory or it may be prolonged. Only in certain instances can microorganisms be established in a manner comparable with that accomplished with insect parasites and predators. Most applications of microorganisms are made in a manner analogous to the application of insecticides. Furthermore, natural outbreaks of disease are usually of a fulminating nature, and of comparatively short duration although the pathogen may be present in the population between epizootic periods. In general, it is difficult to establish pathogens in areas to the extent this is possible with certain insect parasites, although in some instances this has been successfully accomplished.

If the "economic level" of the host density is considerably lower than the "threshold level" of the disease, then control will be temporary and act like chemical control measures. If the economic level of the host density is higher than the threshold level of the disease, then the control can be just as permanent as that accomplished by parasites and predators. Of course, the threshold of the disease may be modified by the physical factors of the environment. It appears that ordinarily disease will give only temporary control and, from a practical standpoint, we must, in these cases, devise means of lowering the thresholds (as has been done in a number of instances). In some cases, however, pathogenic microorganisms may give

prolonged or "permanent" control, as in milky disease of the Japanese beetle; but on the basis of data now available these instances appear to be exceptional.

5. The effect of disease on other density-dependent control factors, such as insect parasites and predators, may be of a direct or indirect kind. At times, but rarely, a pathogen may be capable of infecting not only a given host insect but the host's parasites and predators as well. The pathogen may indirectly affect the parasites and predators by reducing the host population to a point where these agents are unable to find enough hosts on which to breed or on which to feed. In numerous instances, however, disease may greatly reduce the numbers of insects in a population without seriously disturbing the over-all effectiveness of the parasites and predators. It would appear that disease usually has a higher threshold of operation than do insect parasites in a given population.

6. The mode of transmission or dissemination of a pathogen (or its capacity to spread) is of considerable importance in the epizootiology of disease in insect populations. Dissemination of pathogens may occur through the mobility of the microorganism itself, by the activities and movements of the host insects (including such mechanisms as transovarial transmission), by insect parasites and predators, by such physical agencies as wind, rain, and irrigation water, and by man. In general, however, microorganisms are relatively passive and have low degrees of mobility compared with parasites and predators with their searching power. On the other hand, microbial agents may have marked powers of survival and capacity to spread.

APPENDIX

Prior to and during the preparation of this paper the author benefited from copious discussion and disputation of the subject matter with colleagues and associates. Without holding them responsible in any way for any of the views expressed herein, I do wish gratefully to acknowledge the help and counsel of these individuals as well as their generous assistance in critically reading the manuscript: F. T. Bird, E. C. Clark, C. P. Clausen, I. M. Hall, H. S. Smith, R. F. Smith, M. E. Martignoni, and C. G. Thompson.

In the original draft of this manuscript, the author included in the introduction a short section relating to the types of relationships existing between microorganisms and insects. This was done because it was thought wise, before dealing specifically with the influences of microbial disease on insects, to consider briefly the different ways in which insects and microorganisms may be associated when considered from an ecological viewpoint. Moreover, the author is aware of no similar succinct classification of these relationships in the literature. To most biologists the best-known association between microorganisms and insects is that in which the insect serves as a carrier or a vector of a microorganism pathogenic for man or other animals, or for plants. Perhaps the next most familiar insect-microbe relationship is that existing between insects and those microorganisms pathogenic to them. The early studies of Pasteur on the diseases of the silkworm and the beekeeper's familiarity with brood diseases did much to highlight this type of association. Beyond this point (except for the mutualistic role of protozoa in termites) the ecological relationships between insects and microorganisms are not generally recognized or appreciated.

In reviewing the manuscript, some of the individuals named above felt that the citation of these relationships was not germane to the subject matter of this paper, while others thought it constituted an appropriate part of the introductory statement and assisted in an orientation of the reader with respect to the different ways in which populations of microorganisms and insects are interrelated. This was felt to be true even though the present paper is concerned chiefly with but one of several biological relationships known to exist between insects and microbial life. For purposes of simplification, these relationships are presented more in terms of the individual insect's relation to a microbial population than in terms of large populations of insects. In most cases, however, the extrapolation of the principles involved over to a basis of larger insect populations is perhaps obvious. For these reasons it was thought best to remove this section from the body of the paper and incorporate it in an appendix.

Types of Relationships Existing Between Microorganisms and Insects

The various relationships existing between microorganisms and insects may be arranged in a number of categories depending largely upon the narrowness of the boundaries or the degree of specialization one chooses to depict. Basically, however, virtually all the ecological relationships known to exist between these two forms of life may be placed in one of the following nine arbitrary categories:

1. **Insect feeding on substrate previously broken down or changed by the activity of one or more microorganisms.** Obviously this relationship is a rather indirect or remote one, but it is nevertheless a situation in which the activities of one (the microorganism) eventually affects the activities and livelihood of the other (the insect).

An example of this type of relationship between microorganisms and insects is that in which yeasts bring about the fermentation of grapes, thus providing optimum conditions for the developing larvae of drosophila flies (which also feed on the yeasts). A similar relationship may be considered to exist between microorganisms, especially bacteria, and insects that breed in and frequent filth and decaying organic matter. In this general category might also be included the role of microorganisms in reducing the amount of dissolved oxygen in natural waters to serve as a stimulus to the hatching of the eggs of certain mosquitoes.

2. **Free-living microorganisms, especially bacteria and yeasts, serving directly as food for insects.** Noteworthy examples of this category of insect-microbe relationships

include certain mosquito and fly larvae that feed on bacteria, and *Drosophila* flies that ingest yeasts along with the substrate on which they are feeding.

3. Insect and microorganism existing separately but in a more-or-less common or regular association. Insect acts as carrier or intimate host only occasionally or to ensure continuation of relationship or when microorganism is ingested as food. In a sense, this is a specialized extension of the relationship designated under No. 2. Examples of this type of relationship, which would be classified under disjunctive symbiosis, are the fungus-growing ants, termites, and beetles, and the fungi they cultivate. This external association between the fungi and insects is one that provides the latter with certain of their food requirements. The fungi are cultivated in special beds or gardens carefully prepared and maintained by the insects. Each species of insect cultivates only one species of fungus, and only the most closely allied species of insects cultivate the same fungus species. The point requiring emphasis here is the fact that the fungi grow, develop, and derive their nourishment not in or on the insect itself, but in a location separate from it. The only time the fungus or parts of it are removed from their site of development and intimately associated with the insect is when the insect feeds on it or when, as in the case of the attine ants, reproductive bits (for example, a mass of hyphae) are transferred by the virgin queen, by means of her infrabuccal pouch, to a new location.

4. Insect as a host to an adventitious microorganism fortuitously present in or on the insect. In the course of their movements and general activities, most insects, quite by accident, acquire microorganisms of one kind or another. Usually such acquisitions are of a temporary nature and in no sense do they represent the animal's "characteristic" microbiota. To be sure, the types of microorganisms acquired, the frequency with which they are acquired, and the numbers by which they are present in or on the insect depend largely upon the arthropod's environment and habitat. The important aspect of the relationship, however, is that these adventitious microorganisms have no specific significance as far as their relation to the insect is concerned. They are not species ordinarily or regularly found associated with the insect and their presence can be explained only on the basis of fortuitous contact with the insect. Thus, it is not uncommon to find spore-forming bacilli in or on insects living in the soil or on low-growing vegetation. Such bacteria are characteristically present in the soil but may be present in or on the insect only by chance.

5. Insect as a host to commensal microorganisms commonly found associated with them. Admittedly, this category is not far removed from the preceding one. Nevertheless, in studying the microbiota of insects one is impressed by the fact that, like most animals, most species of insects regularly harbor or contain a fairly distinct microbiota. In some instances certain species of microorganisms are found in a given insect species no matter where the insect is found. It is clear that the nature of the commensal microbiota in any particular insect depends a great deal upon the animal's environment and habitat, as in the previous category. In the relationship referred to here, however, the association is usually rather constant and characteristic. In some instances the microorganisms regularly found in the insect may also be found in the animal's environment. Insects such as the housefly and cockroach, for instance, may have upon their body surfaces, as well as in their intestinal tracts, bacteria and other microorganisms usually found in areas of filth and decomposing organic matter. In a sense, such microorganisms are adventitious as far as the insect is concerned, but when the latter spends most of its life in such an environment the associated microflora may be regularly present and characteristic of the insect in that particular environment. In other instances, certain microorganisms are nearly always associated with a particular insect even though other microorganisms for a time become a prominent part of the microbiota. As *Escherichia coli* is a commensal constantly present in the intestinal tract of a healthy man, so are certain species of microorganisms constantly present as commensals in the alimentary tracts of insects, such as the coccuss regularly found in the alfalfa caterpillar.

6. Insect as a vector of microorganisms pathogenic to animals or to plants. As we have already mentioned, the relationships between microbial agents pathogenic for animals or plants and their insect vectors are undoubtedly the best known of all insect-microbe associations. Classic examples are the mosquito transmission of the agents of malaria and yellow fever, as well as the leafhopper transmission of the virus of curly top and beetle transmission of bacterium of cucurbit wilt.

In the case of organisms pathogenic for animals and man, insects are known to transmit bacteria, viruses, protozoa, and nematodes. The biological relationships between these agents and their insect vectors vary considerably in detail, but of primary interest is the fact that in some instances the invertebrate host is merely a mechanical conveyor of the pathogen while in other instances further development, multiplication, or maturation of the agent take place in the insect. A similar statement may be made in relation to the pathogens (bacteria, fungi, viruses, and protozoa) transmitted by insects to plants. The affluence of literature on the role of insects as vectors of disease agents obviates the necessity of presenting further examples of the type of relationship.

7. Insect as a host to extracellular symbiotes (that is, mutualists). Living freely in the lumen of the alimentary tracts or in the associated caeca of many insects are microorganisms (principally bacteria and protozoa) that are known to exert a beneficial effect on the life processes of their host. In many instances the beneficial role played by the microorganisms is unknown, or only assumed, but most of these cases have had only superficial study.

A classic example of the type of relationship referred to here is that existing between termites and their rich protozoan fauna. Over 500 species of termites have been studied in this regard and approximately 300 species of protozoa have been described from these hosts. The termites are dependent upon the protozoa for the digestion of the wood upon which the insects feed, making the cellulose contained in the wood available to the termites. A similar relationship prevails between the wood-eating roach (*Cryptocercus*) and its protozoan fauna.

In some insects bacteria play an active part in the digestive processes of the host. Thus, the alimentary tract of larvae of lamellicorn beetles has special pouches or "fermentation chambers" in which cellulose is broken down enzymatically by bacteria making it more easily assimilated by the insect. Not so clearly defined is the function performed by the bacteria living in the "gastric caeca" of the higher Hemiptera. These bacteria are morphologically characteristic of the species of insect harboring them, and they pass from generation to generation in association with the egg.

8. Insect as a host to intracellular symbiotes (that is, mutualists). The tissue cells of many normal insects and ticks harbor specific living microorganisms usually bacterial, rickettsial, or yeastlike in nature. The intracellular symbiotes frequently occur in the cells of special organs known as "mycetomes" attached to the wall of the alimentary tract or located at various other parts of the body. The symbiotes are transmitted from one generation of the host to the next, usually via the egg. As far as their function is concerned, it is known that in some insects the symbiotes produce vitamins upon which the life of the insect is dependent. Other growth-promoting or growth-depressing substances may also be involved. In other insects it is believed that they produce hormones or hormone-like substances that benefit the host. In still other insects it appears that the symbiotes fix atmospheric nitrogen required by the host. In any case, the insect-symbiote relationship is an intimate one since every individual of an insect species harbors the symbiote specific for that species.

The arthropod hosts of these intracellular symbiotes occur in most of the principal orders of insects and numerous families of ticks and mites. Insects of both medical and agricultural importance are concerned, including such groups as cockroaches, lice, bedbugs, reduviid bugs, aphids, scale insects, beetles, ants, and many others.

9. Insect as a definitive host of microbial agents to which it is susceptible. In other words, the microbe-insect relationship may be that in which the microbe is a pathogen whose activity causes disease and frequently death in insect host.

The principal groups of microbial agents responsible for infectious diseases in insects are: viruses, bacteria, fungi, protozoa, and nematodes. Examples of diseases caused by these agents abound (see Steinhaus, 1949), and rapid strides are being made in accumulating knowledge concerning them. The regular or periodic occurrence of disease among insect populations itself constitutes an ecological factor of great importance from the practical standpoint as well as from that of insect ecology generally. The insect ecologist in particular should not lose sight of the fact that infectious disease is a manifestation of parasitism. It represents the reaction of the insect to invasion of the animal's tissues by a microparasite. It is simply a form of the struggle of living beings for food, shelter, and propagation as expressed in a host-parasite relationship.

Of the nine insect-microorganism associations we have delineated, it is the one last named with which we have been primarily concerned in the present paper.

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NOTE: The present paper was already in page proof at the time of the appearance of the book by H. G. Andrewartha and L. C. Birch, *The Distribution and Abundance of Animals* (University of Chicago Press, 1954, 782 pp.). Accordingly, the principles discussed in this paper have not been dealt with in the light of some of the beliefs and conclusions (for example, those pertaining to the distinctions between density-dependent and density-independent factors) arrived at by these authors.

